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EDITORIAL

THE PLACE OF ENDODONTICS IN DENTAL PRACTICE

DENTISTRY has had some interesting and exciting episodes in its rather brief history as a profession. The "amalgam war" raged for several years and was the principal cause of dissolution of the first national dental society, the American Society of Dental Surgeons. Briefly, amalgam was introduced into this country by two French charlatans and was a challenging rival to plugged gold fillings. The material was so poor, however, and its correct use was so little understood that those dentists who used it were ousted from membership in dental societies. It remained for such men as J. Foster Flagg to organize a group of unbiased thinkers in the dental profession to test this material both in the laboratory and clinically. They demonstrated that amalgam was a satisfactory filling material for the saving of carious teeth, and amalgam came to be accepted.

A somewhat parallel episode occurred shortly after the turn of this century when the theory of focal infection was promulgated and pulpless teeth were extracted in wholesale fashion. Curiously enough, the initiator of the theory, William Hunter, had never intended that his barbs of criticism should be aimed at the pulpless tooth. Instead, his criticism was directed at periodontal disease caused by faultily designed and constructed crowns and by bridgework around which he had observed accumulations of debris which harbored myriads of microorganisms. When dental x-ray machines became available shortly afterward and disclosed areas of rarefaction around root apices, a veritable orgy of extraction of pulpless teeth occurred. The treatment of pulpless teeth then fell into disrepute. It was forgotten that such teeth had been treated with "eyes closed," as they had not had the benefit of the roentgenogram for diagnosis prior to or during treatment. At the time the focal infection theory was developed there probably were not more than a dozen x-ray machines in use by dentists in this country, according to Dr. Howard R. Raper. Several dental schools discontinued not only the treatment of pulpless teeth in their clinics but the teaching of the subject as well.

Today we are witnessing a renascence of interest in endodontic practice, such as has never occurred before in dental history. Not only is endodontics

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being taught in every dental school in this country but also it is probably being practiced to a greater degree than ever before in this century. What is the place of endodontics in dental practice today?

A review of the program of the American Dental Association's centennial meeting shows that an entire day was devoted to a forum on endodontics, an entire afternoon was devoted to clinical lectures, a color television clinic on endodontic practice was given, and in addition a number of table clinics and motion picture films were presented. Similarly, a review of the program of the recent Greater New York meeting discloses the following program in endodontics: five projected clinics, an afternoon forum, a clinical demonstration, a special clinic, four registered clinics, and six motion picture films. Furthermore, attendance at these sessions has reached capacity or beyond in most cases.

The interest shown in endodontics by the general practitioner is a reaffirmation that the goal of the dentist is not to "extract and replace" but rather to "conserve and restore" the natural organs of the mouth. Endodontics has won back its rightful place in dental practice.

OPERATIVE ORAL SURGERY

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UNILATERAL HYPERPLASIA AND EXOSTOSIS OF THE MANDIBULAR CONDYLE

A Clinical Syndrome

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UNILATERAL facial asymmetry resulting from enlargement of the mandibular condyle is a well-known clinical entity.[1-8, 11, 13, 14, 16]

Chondromas, exostoses, hyperplastic deformities, and malignant tumors of the condyle may produce facial asymmetry. However, few detailed histologic reports of these entities as they involve the mandibular condyle are available. The present report deals with a case of unilateral facial asymmetry resulting from hyperplasia of the mandibular condyle and an associated exostosis; a definitive clinical syndrome is described.

CASE REPORT

Patient T. H., a 26-year-old white woman, presented to the Oral Surgery Service, Research and Educational Hospitals, on Feb. 8, 1955, for correction of marked facial asymmetry and malocclusion.

Onset and Course.—The patient stated that when she was 10 years of age she fell and hit her face against a door, and a cut on the chin resulted. No therapy was given. The function of the jaws remained normal.

At the age of 15 the patient received a fist blow on the left side of the mandible while at play. There was some soreness, but no functional difficulty was experienced. About a year later the patient noticed that her chin seemed more prominent on the right side and that the left side of her face was flat. This deformity became progressively worse, and for the past three years the patient had been extremely self-conscious about her appearance. She had also noticed a progressive speech impediment and difficulty in articulation.

The past medical history was noncontributory.

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Presented before the Oral and Maxillofacial Surgery Seminar on Oct. 26, 1955.

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Clinical Findings.—Clinical examination revealed a well-nourished woman with marked asymmetry of the face (Figs. 1 to 4). This asymmetry was characterized by a flatness on the left side, prognathism with deviation of the chin to the right side, and increased height on the left ramus. There was marked prominence of the left malar region anteriorly. No palpable enlargements were present in the regions of the temporomandibular joints. The patient could open and close her mouth with no evidence of clicking or grating. Lateral movements were slightly restricted. A small scar was present on the chin.

Fig. 1. Fig. 2.

Fig. 3. Fig. 4.

Figs. 1 to 4.—Photographs showing marked facial asymmetry of the face of a 26-year-old woman. Note the flatness of the left side of the face, prognathism with deviation of the chin to the right side, increased height of the left ramus, and marked cross-bite.

Intraorally, there was a prognathic relationship between the jaws, with a marked cross-bite. The buccal cusps of the right upper molars occluded in the central fossae of the right lower molars. The maxilla appeared to be somewhat underdeveloped. The palatal vault was high and narrow. Soft tissues were essentially normal. The ears, nose, and throat were negative. There was no evidence of deformity of the temporal bones.

The disorder was tentatively diagnosed as (1) unilateral hyperplasia of the left mandibular condyle and (2) Class III malocclusion with cross-bite and open-bite.

Roentgenographic Findings.—Roentgenograms of the temporomandibular joints revealed normal configuration of the right side. On the left side, however, there was a narrowing of the articular condyle. The temporal articulation was flattened and sclerotic, and the articular tubercle was poorly defined and somewhat flattened. Lateral and anteroposterior projections

of the mandible revealed a disproportion between the two sides. The left side of the mandible had a greater ramus height and a longer body length. The coronoid process was undersized (Figs. 5 and 6).

The condition seen in the roentgenograms was interpreted as asymmetry of the mandible with apparent enlargement of the left mandible and condylar process. This had the appearance of a benign process, and it was thought that it probably represented an osteoma.

Laboratory Data.—Routine hematologic tests were within normal limits. Serologic tests were negative. Urinalysis was normal. Blood calcium, phosphorus, and alkaline phosphatase levels were normal.

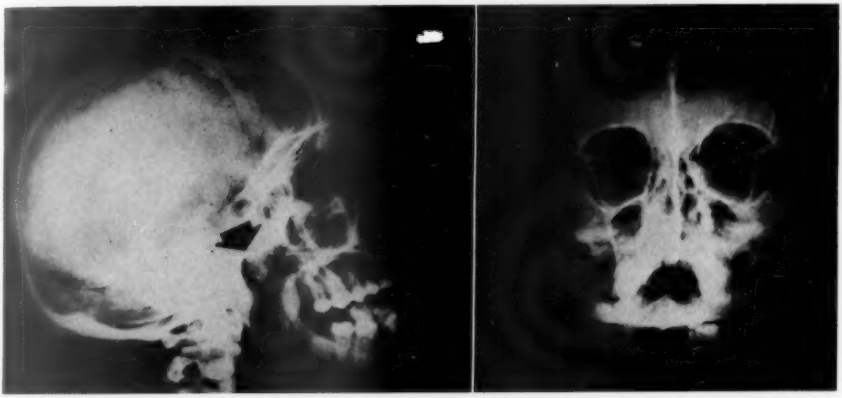

Fig. 5. Fig. 6.

Figs. 5 and 6.—Roentgenograms showing lateral and anteroposterior views of the mandible. Note that the left side of the mandible has a greater ramus and a longer body length than the right side. Also notice the enlarged condylar tumor (arrow) of the mandible.

Treatment Planning.—Plaster casts were made of the teeth. It was possible, by manipulation of the casts, to restore the occlusion to about normal. In view of the possibility of favorable restoration of occlusion, as indicated by the casts, resection of the enlarged condyle and a cosmetic restoration were decided upon.

Hospital Course.—On May 12, 1955, under local anesthesia, mandibular and maxillary arch bars were applied to the teeth for postoperative intermaxillary fixation. On May 13, 1955, the patient was taken to the main operating room where, after induction of nasotracheal anesthesia, a condylectomy of the left side was performed via the preauricular approach. At operation, there was no evidence of erosion of the temporomandibular joint.

On the second postoperative day, the patient had a temperature of 101° F. and edema of the left eyelids. She complained of a sore throat. Edema was marked on the third day and involved the right side of the face also. There was no affection of the seventh nerve. Dressings were removed on the fourth day, and by the eighth day the edema had subsided. Sutures were removed on the tenth postoperative day; rubber elastics were applied to bring the occlusion into the desired position.

The patient was discharged from the hospital on May 24, 1955, and was followed in the outpatient clinic. After four weeks, the intermaxillary elastics were removed and the patient was allowed to exercise her jaws.

There was a remarkable change in the patient's speech, appearance, and occlusion (Fig. 7). The chin had been brought to the center, and masticatory function was greatly improved. The patient was followed as an outpatient for two years and tentatively dismissed.

Pathologic Findings.—

Gross description: The formalin-fixed specimen consisted of the left mandibular condyle with an attached bony mass on its anterior surface. It measured 3.2 by 3.5 by 2.5 cm. The condylar head was covered by a thin, translucent membrane. Most of the lateral surface

Fig. 7.—Intraoral photographs showing occlusion four weeks following surgical removal of the condyle. (Compare with Fig. 4.)

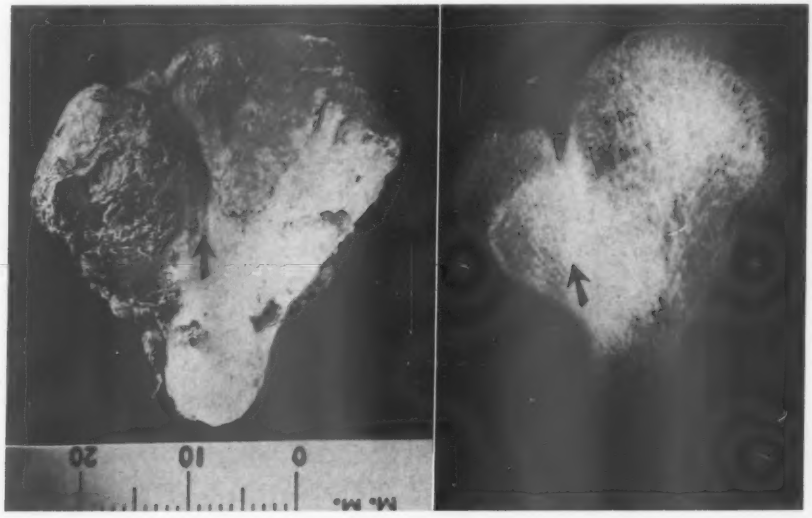

Fig. 8. Fig. 9.

Fig. 8.—Lateral view of surgical specimen of resected left mandibular condyle with attached bony mass on its anterior surface.
Fig. 9.—X-ray view of the gross specimen showing sclerosis at the area of attachment of the bony mass to the condyle.

was denuded. However, soft tissue and muscles were attached to the anterior and mesial surfaces. There was an irregular bony growth anteroinferior to the condylar head. It was attached with the head at the neck of the condylar process (Fig. 8). An x-ray film of the specimen showed sclerosis at the area of attachment of the bony mass to the condyle (Fig. 9).

Microscopic description: The condyle was sectioned in the sagittal anteroposterior direction (Fig. 10). The condyle was found to be enlarged, and an accessory bony growth was attached to its anterior surface. Both the condyle and the attached growth consisted of lamellated bone tissue which formed an outer, rather thin compacta and a spongiosa of relatively thick trabeculae. The compacta showed few cross sections of Haversian canals. The bone marrow of both the condyle and the growth was of the hematopoietic type in the two-thirds toward the ramus and of the adipose type in the third facing the joint.

Fig. 10.—Histologic section through gross specimen. Note the enlargement of the condyle and accessory bony growth (*BG*) attached to its anterior surface. Note that the condyle and the growth have hematopoietic bone marrow facing the ramus (*R*) and adipose bone marrow facing the joint. (Magnification, ×5; reduced ¼.)

The joint surface of the condyle was covered by a thin layer of fibrous tissue. Between this layer and the bony structures was a layer of hyaline cartilage. The chondrocytes appeared in small and large groups which were separated by septa of ground substance (Fig. 12). A definite calcified layer of cartilage was missing. There was no limiting bony plate between spongiosa and cartilage (Fig. 11). Instead, the bone marrow projected into the cartilage. These projections of the bone marrow were of a fibrous nature. In some places chondroclasts lined the surface of the cartilage; in others, it was lined by thin layers of bone tissue. The anterior surface of the condyle was covered by periosteum. In the deeper

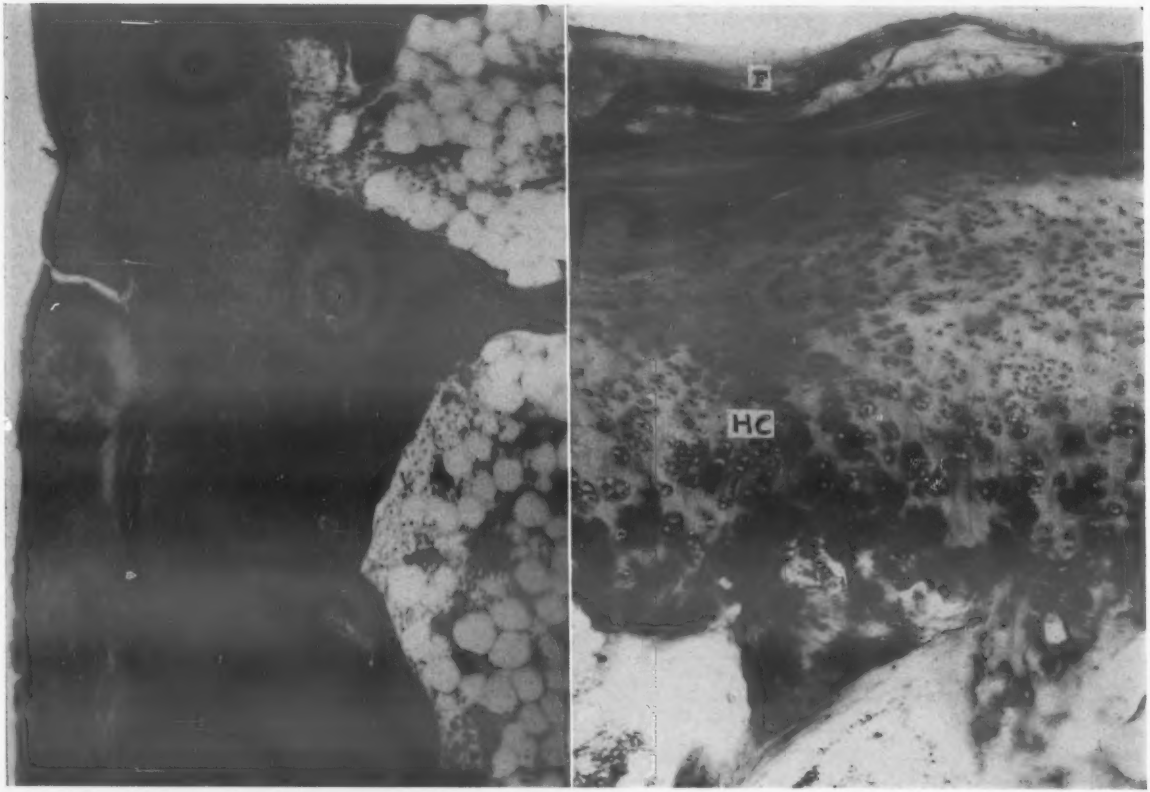

Fig. 11. Fig. 12.

Fig. 11.—Photomicrograph of area *A* in Fig. 10 showing joint surface and bone marrow of the adipose type. Note absence of a limiting bony plate between spongiosa and cartilage. (Magnification, ×150; reduced ¼.)

Fig. 12.—Photomicrograph of area *B* in Fig. 10. The joint surface is covered by a thin layer of fibrous tissue (*F*). Between this layer and the bony structures is a layer of hyaline cartilage (*HC*). Note small and large groups of chondrocytes. Note, too, that the bone marrow projects into the cartilage. (Magnification, ×150; reduced ¼.)

Fig. 13.

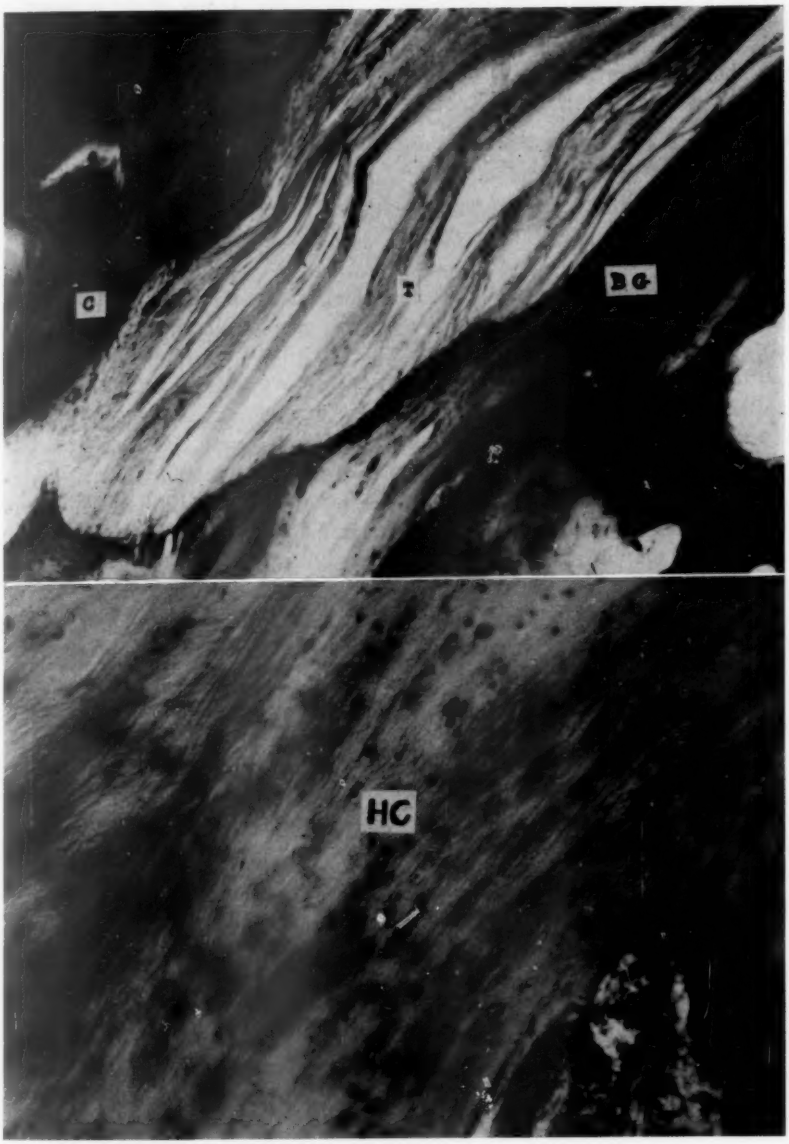

Fig. 14.

Fig. 13.—Photomicrograph of area *C* in Fig. 10. Note the attachment of the tendon (*T*) on the bony structure of the condyle (*C*) and on the accessory bony structure (*B*). (Magnification, ×150; reduced ¼.)

Fig. 14.—Photomicrograph of accessory bone growth, area *D* in Fig. 10. Note the chondrocytes in the island of hyaline cartilage. (Magnification, ×150; reduced ¼.)

layers this periosteum was fused with a sheet of fibrocartilage which separated the condyle from the accessory growth in its cranial part. The caudal portion of the accessory bony growth was fused with the bony portion of the condyle without any sharp border. In other words, the fibrocartilaginous portion of the tendons inserted directly on the bony structures (Fig. 13). No sign of growth could be observed. At the anterior surface of the accessory growth two islands of hyaline cartilage, separated by connective tissue, appeared as additional growth centers (Fig. 14). In the center of the islands connective tissue was seen adjacent to the surface of calcified hyaline cartilage. Some surfaces were lined by a thin layer of bone tissues.

Diagnosis:* Hyperplasia and exostosis of the mandibular condyle.

DISCUSSION

Facial asymmetry resulting from enlargement of the condyle, though a well-known clinical entity, is still of relatively rare occurrence. Fourteen cases were recorded from the literature in 1926.[3] By 1946 the total number of cases reported had grown to thirty.[6] Several cases have been added recently.[5, 14, 15]

Facial asymmetry can also result from (1) removal of the condyle during the period of active growth,[8] (2) lack of condylar growth, (3) forward dislocation of the condyle, and (4) premature loss of posterior teeth.[9]

The factors which produce enlargement of the mandibular condyle and, in turn, facial asymmetry are quite variable. Condylar hyperplasia is one[7]; exostosis is another.[11] It is considered unusual to find exostosis associated with hyperplasia of the condyle.[11]

The etiology of condylar hyperplasia is not clear. Trauma and inflammation may play a role as either initiating or predisposing factors.[9] In this case, there was a history of trauma some sixteen years before the deviation of the chin was observed. In one of the cases presented by Gruca and Meisels,[3] the patient also gave a history of trauma. This was considered to be noncontributory. However, Gruca and Meisels[3] cited another case in which chronic middle ear infection was associated with mandibular condyle enlargement.

Exostosis is one of eight primary tumors of the condyle, any one of which is capable of producing facial asymmetry. Usually exostosis is found on otherwise normal condyles or is associated with osteoma of the condyle[11]; it may present at the site of insertion of the external pterygoid muscle.[11]

Regardless of the etiological factors, Vazirani (one of the authors) contends that unilateral enlargement of the condyloid process of the mandible produces a definitive clinical syndrome which includes the following:

(1) Little or no interference with motion of the jaw.

(2) Absence of pain in the very early stages, with intermittent and occasionally severe pain in the later stages. Noise in the joint on movement.

(3) Slowly progressive vertical elongation of one side of the face produced by an increase in the height of the ascending ramus of the mandible.

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*By T. E. Bolden and Joseph P. Weinmann.

(4) Marked prognathism with deviation of the chin to the opposite side and downward.

(5) Mild or marked malocclusion on the affected side, usually associated with cross-bite and open-bite.

(6) A progressive speech impediment and masticatory dysfunction.

The entity occurs in males and females almost equally, and it apparently is initiated over a very wide age range.

Condylectomy, the accepted form of therapy, is believed to have been initiated by Eckert[2] in 1899.

SUMMARY

A case of unilateral hyperplasia and exostosis of the mandibular condyle is presented. This benign lesion produced marked facial asymmetry and speech impediment in a 26-year-old woman.

The etiological factors responsible for enlargement of the mandibular condyle are discussed briefly. A detailed histologic study of the resected condyle is presented, and a definitive clinical syndrome is described.

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CHOICE OF ANTI-INFLAMMATORY AGENTS IN ROOT RESECTION

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OTHER investigators have reported that pre- and postoperative administration of certain antihistaminics and hydrocortisone acetate reduces the swelling or edema associated with oral surgical procedures.[1-6] Blinderman[1] has found in a series of 150 patients treated for impacted third molars that, of those receiving 12.5 or 25 mg. promethazine hydrochloride thirty minutes preoperatively, less than 10 per cent later developed edema. In a smaller series, Moss[2] reached similar conclusions. Keesling,[3] on the other hand, has reported no significant reduction in postoperative edema, pain, or healing after giving 6.25 mg. promethazine hydrochloride every four hours during the day preceding surgery.

Microcrystalline cortisone acetate given parenterally produces a blood steroid level that is sustained three to four times longer than similar doses of cortisone given orally. The extended activity of the parenteral form offers theoretical advantages over orally administered steroid in the prevention of edema. Promethazine hydrochloride given orally has longer action than similar parenteral doses. This experiment was designed to test the effectiveness of these longer-acting agents in controlling edema when administered only on the day of surgery.

Study Groups and Medication.—Four study groups, totaling 228 patients, were established:

1. Fifty-two patients received no medication.
2. Eighty-one patients received one 25 mg. tablet of promethazine hydrochloride* one hour before surgery and a second tablet on retiring the same night.
3. Twenty-four patients received 50 mg. of cortisone acetate intramuscularly one hour before surgery.
4. Seventy-one patients received 50 mg. of cortisone acetate intramuscularly plus one 25 mg. tablet of promethazine hydrochloride one hour before surgery; a second 25 mg. tablet was given on retiring.

CLINICAL METHODS

All patients in the four groups were subjected to root canal preparation, irrigation, and filling performed under a rubber dam. After removal of the

*Promethazine hydrochloride is available as Phenergan hydrochloride from Wyeth Laboratories, Inc., Philadelphia, Pennsylvania.

dam, apicoectomy or periapical curettage was done. A horizontal incision in the attached gingiva was extended to the labial roots of the proximal teeth. A slight vertical extension at each end of the incision permitted formation of a mucoperiosteal flap.

Preoperative and Postoperative Care.—Analgesic medication was restricted to tablets containing acetylsalicylic acid, caffeine, acetophenetidin, and codeine phosphate. Postoperatively, 107 patients received analgesics. A number of patients also employed ice packs in a controlled study.

Four patients received antibiotics preoperatively and postoperatively when an acute apical reaction did not respond to drainage through the root canal.

Method of Observation and Evaluation.—Absence of edema was verified in each patient before and immediately after the surgical procedure. A postoperative examination was made twenty-four hours later, or as soon as possible when the one-day follow-up proved unfeasible.

Criteria for evaluation of edema were as follows:

1. *None*—no perceptible change in lip contour.
2. *Moderate*—noticeable edema localized to the operative site.
3. *Severe*—considerable edema, most often infiltrating tissues surrounding the operative site.

Additional observations were made on degree of sedation (if any) resulting from medication, side effects, and healing pattern of the surgical incision.

Method of Analysis.—In this study, edema either did or did not exist. Magnitude of edema was disregarded during statistical analysis. Data were analyzed by the Chi square test[7] in simple 2×2 tables.

RESULTS

No significant difference appeared between the incidence of postoperative edema in the control group and in the group receiving only cortisone acetate. Only nine of fifty-two patients (about 15 per cent) given no medication remained free of edema postoperatively. Table I presents patients' responses to drug therapy.

In the present study, the combination of promethazine and parenteral cortisone produced the best results. More than one-half the patients receiving both drugs developed no perceptible edema after the surgical procedure.

Promethazine alone proved slightly less than two-thirds as potent as a combination of the two drugs in reducing incidence of edema. Compared with the control group, this reduction was found significant.

It was observed that six of the patients in Table I who received medications and who subsequently developed facial edema were not aware of this swelling until the second morning after surgery. This occurred after blood levels of the drugs used were presumably greatly reduced. In this group, at least, it is possible that continuation of drug therapy into the second day might have been beneficial.

TABLE I. RESPONSE TO ANTI-INFLAMMATORY THERAPY FOLLOWING IMMEDIATE ROOT RESECTION

MEDICATION	DOSAGE (MG.)	DEGREE OF POSTOPERATIVE SWELLING OR EDEMA				CHI^2 VALUE	PROBABILITY	SIGNIFICANCE
		NONE	MODERATE	SEVERE	TOTAL			
None (control)	—	9	25	18	52	—	—	—
Promethazine hydrochloride	50	26	31	24	81	4.42	$0.025 > P < 0.05$	Significant
Cortisone acetate	50	6	12	6	24	1.18	$0.25 > P < 0.30$	Not significant
Cortisone acetate and promethazine hydrochloride	50 50	37	24	10	71	18.0	0.001	Highly significant
Total responses		78	92	58	228	—	—	—

Most patients became slightly drowsy within one hour following the first 25 mg. tablet of promethazine. No sedation appeared after cortisone administration. Comparison of promethazine-treated patients with the control group indicated that promethazine did not alter the postoperative pain response. In our opinion, however, there was a reduction in the analgesic dosage required for relief of postoperative pain. Neither healing pattern nor healing time was influenced by either drug in this study.

The use of ice packs did not influence postoperative edema, according to a comparison of the study and control groups.

DISCUSSION

Severe inflammation and edema need not be accepted as inevitable postoperative sequelae in apicoectomy. Histamine mobilization and degradation of the mucopolysaccharide ground substance in some tissues are at least partly responsible for these conditions.[8, 9] Recognition of this fact has led to specific antihistaminic chemotherapy serving to reduce inflammation and edema associated with oral surgery.

Both reactions, with attendant increase in capillary permeability, are normal physiologic responses to stress and tissue trauma.[8] In surgical procedures these reactions should be controlled. Without such control, the patient may experience greater discomfort, as well as extended healing time and other postoperative complications.

Among its many actions, cortisone causes a selective decrease in capillary permeability. It also tends to prevent enzymatic degradation of the mucopolysaccharide ground substance. Antihistaminics antagonize histamine and tend to decrease capillary permeability. Both agents thus act to reduce inflammation and edema.

There are other theories to explain the physiologic effect of antihistaminics and cortisone on the human organism.[8-10] However, no one hypothesis fully accounts for the occasional failure of antihistaminics to reduce postoperative edema, as noted by some investigators.[5, 6] Undoubtedly, antihistaminic agents

differ in potency. Differences in dosage, surgical technique, and method of evaluation may explain differences in response observed by some investigators.

Response of patients to promethazine in our study confirms the experiences of Blinderman[1] and Moss.[2] One reason for Keesling's[3] failure to observe efficacy in promethazine could have been dosage schedule. He administered 25 mg. over a twenty-four-hour period, whereas patients in the present study received 25 mg. within one hour of surgery.

Failure of cortisone to reduce edema significantly in this study is only moderately surprising. The anti-inflammatory action of cortisone is noted to be less potent than that of hydrocortisone in equivalent doses. Although other investigators[8] have found that a 50 mg. injection of hydrocortisone has effectively reduced inflammation, 50 mg. of cortisone might easily have been insufficient. In addition, the size of the cortisone-treated group was small.

This was not a double-blind study. Patients were selected for each category of medication as they presented themselves. If medications were contraindicated, the patient was then used as a control. In this study a history of tuberculosis was a contraindication to cortisone therapy, and promethazine was contraindicated when the anticipated drowsiness following its administration would influence the patient's driving.

SUMMARY

Promethazine hydrochloride, parenteral cortisone acetate, and a combination of the two agents were administered to study groups totaling 228 patients undergoing immediate root resection. As compared with findings in a control group, the incidence of postoperative swelling or edema was greatly reduced in patients receiving the combination of drugs. Promethazine proved efficacious, but less so than the two drugs together. The healing pattern was not perceptibly altered. Side effects were absent, except for slight drowsiness produced by promethazine.

ADDENDUM

After completion of this study, a group of thirty-five patients were given one 25 mg. tablet of promethazine hydrochloride and one 5 mg. tablet of Meticorten one hour before surgery. This was repeated at bedtime. Results were only slightly better than those recorded for the promethazine hydrochloride group.

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DENTIGEROUS CYST INVOLVING A SUPERNUMERARY MAXILLARY MOLAR WITH ENCROACHMENT ON THE MAXILLARY ANTRUM

Report of a Case

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THE literature is replete with articles on the presence of supernumerary teeth, their importance and possible relationship to atavism of the adult dentition, and the treatment of various lesions involving such teeth. This case report is being presented not because it is a new addition to the literature but rather because it points up a certain complication that can occur, namely, antral involvement. While many articles have been published on cysts involving mesiodens and the so-called fourth molar, little has been written about those encroaching on the maxillary antra. It is our belief that this complication is more common than has been suspected.

Since supernumerary teeth are not an unusual finding in either the maxilla or the mandible, any area of the dentition may present this finding, although the molar regions are somewhat more frequently involved. While it is true that the diagnosis and treatment of lesions of supernumerary teeth do not vary from those utilized in the care of the normal deciduous and permanent dentitions, nevertheless there may arise certain situations which present peculiar problems.

Cystic lesions involving supernumerary teeth are not common. The third molar areas present possibly the most common location for cystic degeneration of the follicular sac, followed closely by the anterior maxillary region. The greatest problem that these cystic degenerations can present, aside from possible mutation into ameloblastoma or carcinoma, is encroachment on the maxillary antra. This is a serious complication, requiring the utmost caution and care in diagnosis and treatment. Even under the protection of antibiotic therapy, there may arise difficulties which preclude early healing and lead to a protracted recovery. It behooves the diagnostician to evaluate this

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situation from every conceivable standpoint before arriving at a decision that proclaims antral involvement. The case to be presented bears out this argument.

CASE REPORT

The patient, a 22-year-old male Negro inmate of the Federal Reformatory in Petersburg, Virginia, first presented himself at the Reformatory Dental Clinic on May 27, 1957, where he was seen by one of us (L. L.). He had a chief complaint of "drainage and tenderness" in the region of the upper right molar and premolar teeth. This picture of drainage and tenderness had persisted for several days and was increasing somewhat in intensity.

The past dental history was not essentially significant. Apparently adequate dental care had been undertaken during his adolescence, and except for having had the upper right second premolar extracted some months before he had never experienced dental difficulty. The medical history was also without significance.

Clinical examination at this time showed the dental arches to be well formed, with only a few dental restorations present. The positive findings were limited to the right side of the maxilla in the premolar region, where an apparently poorly healed second premolar socket was observed. The surrounding tissue was not inflamed, but extruding from the socket itself was a small mass of putrescent tissue with some evidence of pus formation. The upper right first molar was quite mobile and exhibited some tenderness on percussion. No swelling was noted either adjacent to the teeth themselves or in the mucobuccal fold. Some tenderness was elicited by pressure over the apical ends of the first molar and over the second premolar socket. After the socket was thoroughly irrigated with warm sterile saline, a probe was inserted into the socket (Fig. 1) to determine its depth and to establish whether or not the maxillary antrum was involved, but resistance was encountered at the apical end of the socket.

No lymphadenopathy or other extraoral findings were observed at this time, although the patient stated that he had felt as though the right cheek were swollen and puffy during the first days of his symptoms.

Roentgenographic examination with both periapical and occlusal films (Figs. 2 and 3) revealed the presence of what appeared to be an odontoma-like mass immediately superior to the first molar near the apical end of the second premolar socket. The impression gained from these views was that of an odontoma with probable cystic involvement.

Due to lack of facilities for further study of this lesion, a consultation was obtained with the Chief of the Section of Oral Surgery (H. I. M.) at the nearby Army Dental Clinic at Fort Lee, Virginia, on May 29, 1957. Essentially the same findings were recorded. With the addition of other roentgenographic views, namely Waters' views and lateral projections of the antra (Figs. 4 and 5), the following roentgenographic report was made:

> *Maxillary antra:* Waters' and lateral projections of the maxillary antra demonstrate the antra to be well-developed, clear, and well-aerated except for the presence of a triangular opaque density situated within the alveolar sulcus of the right maxillary antrum. The appearance of this object has the appearance of an incompletely developed tooth, possibly a tricuspid. As far as can be visualized on these film studies, it appears to be sitting entirely within the boundaries of the right maxillary antrum. Appropriate dental x-ray studies of the right superior alveolar ridge would serve for better evaluation of this area. The maxillary antra are otherwise negative.
>
> *Captain Edenfield, M.C.*

From this tentative summary, an initial diagnosis of a dentigerous cyst involving a supernumerary maxillary molar tooth with subsequent compression, but not involvement, of the right antrum of Highmore was entertained. It was our feeling that the antrum was not involved but only encroached upon, because of the clearness, good aeration, and lack of definitive symptomatology involving the antrum. The original dental studies seemed to corroborate this opinion.

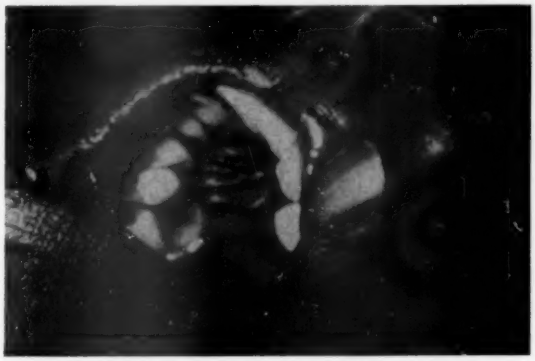

Fig. 1.—Photograph showing the dimpling of the alveolar ridge in the area of the extraction with a silver probe in the position of the missing tooth.

Fig. 2.

Fig. 3.

Fig. 2.—Periapical roentgenogram showing what appeared to be an odontoma-like mass in the area of the missing second premolar.

Fig. 3.—Occlusal roentgenogram of the area shown in Fig. 2.

Since no apparent systemic involvement was noted, and because of the difficulty of arranging immediate surgical intervention due to the stringent regulations of the Federal Reformatory, it was decided that the patient should be placed on a regime of systemic prophylactic procaine penicillin G, 600,000 units daily, until June 4, 1957, when arrangements were made for the necessary surgery. Along with this, warm saline irrigations were encouraged to maintain cleanliness of the involved area.

Fig. 4.

Fig. 5.

Fig. 4.—Waters' view of the antra showing the clarity, aeration, and development of the antra and the position of the lesion *(arrow)* in relation to the right antrum.

Fig. 5.—Lateral projection of the right maxillary antrum showing the over-all relation between the dentigerous cyst containing the supernumerary tooth *(arrow)* and the surrounding structures.

On June 4, 1957, the patient was premedicated with 100 mg. of secobarbital (Seconal) orally, and the right infraorbital and posterior superior alveolar nerves were blocked with 2 per cent lidocaine hydrochloride (Xylocaine) containing 1:100,000 epinephrine. A mucoperiosteal flap was then reflected along the buccal aspect from the upper right cuspid to the upper right second molar. Upon reflection of this flap, a small area of perforation of the buccal plate of bone was noted well above the socket of the previously extracted second premolar. With rongeurs, the thin overlying bone was removed, revealing a bluish gray, thickened sac of tissue that extended from the area of the apex of the first premolar to the area of the first molar. This sac was freed as much as possible with curettes, but it could not be entirely separated in this manner due to the extreme tenaciousness of the membrane. The supernumerary tooth was then easily elevated and removed, and the entire space was carefully curetted. A small opening into the right antrum was noted approximately at the former site of the apex of the supernumerary tooth (Fig. 6), but no other antral involvement was observed. The apex of the upper right first premolar was seen to have not been denuded by this cyst, and this tooth was left in position. The entire antrum had been displaced superomedially by the cystic expansion. The first molar was removed because of the loss of supporting bone and the infringement of the lesion on the apices of its roots (Fig. 7).

Fig. 6.

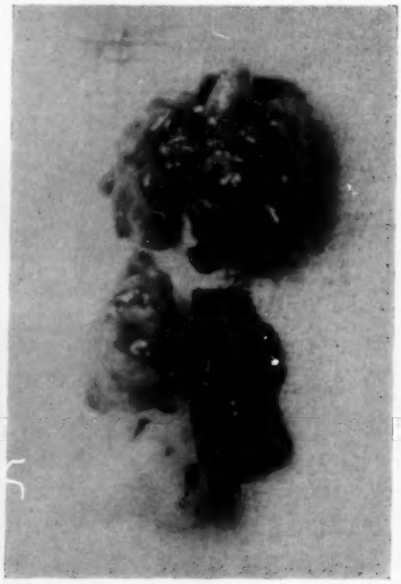

Fig. 7.

Fig. 6.—Operative photograph showing the small antral perforation at the site of the apex of the supernumerary tooth.

Fig. 7.—The gross specimen showing the proximity of the cyst and its contained tooth to the permanent first molar tooth.

The bony margins were filed smooth and a petrolatum impregnated gauze dressing was placed lightly into the cavity to cover the exposed bony confines. The mucoperiosteal flap was carefully coapted with 000 black silk sutures, and the patient was instructed to place a pressure dressing to obtain hemostasis.

The patient was then returned to the Reformatory Infirmary, where he was placed on procaine penicillin G, 600,000 units daily intramuscularly, and streptomycin, 0.5 gram daily

intramuscularly. Codeine, 0.5 grain by mouth every four hours if needed for pain, was prescribed, and ice packs were applied to the affected area to prevent undue edema. The patient spent a comfortable night without taking any analgesics, and on the first postoperative day he exhibited minimal swelling. The same antibiotic therapy was maintained until June 8, 1957, when the streptomycin was discontinued. Penicillin was discontinued on June 10, 1957.

Fig. 8.—Postoperative lateral projection showing essentially the same area as in Fig. 5.

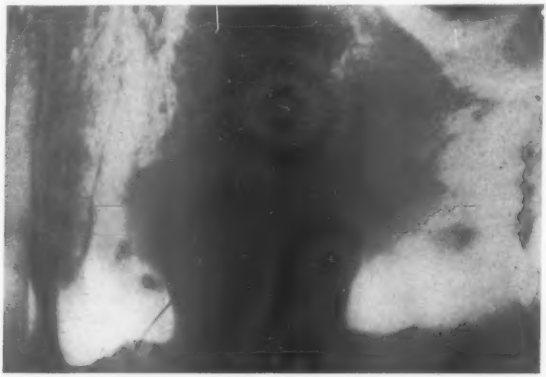

Fig. 9.—Postoperative periapical roentgenogram taken two months later showing good bony regeneration and healing.

Recovery was rapid and healing proceeded well. Postoperative roentgenograms revealed the beginning of bony healing (Figs. 8 and 9).

The pathologic description which follows is quoted verbatim from the report of the New York Institute of Clinical Oral Pathology:

Gross: The crown of the upper first molar is intact, as are the buccal and palatal roots. A small amount of buff soft tissue is present in the interradicular space.

The supernumerary tooth is deformed. The crown shows destruction and is filled with a dark-gray to black material. A curved root measures 9 mm. in length. At the neck buff soft tissue is attached.

Two firm elastic irregular tissue particles, buff in color, aggregate 20 × 15 × 8 mm.

Microscopic: In the palatal portion of the first molar pulp chamber there is rather diffuse infiltration by plasma cells in a rather small area. The pulp of the root canals is somewhat condensed and shows fibrillar condensation, as well as calcification. The periodontal soft tissues show nests of resting epithelium and there is moderate infiltration by plasma cells extending to the tissue surrounding the apices.

A section of the supernumerary tooth shows an irregular mass of hard substance made up by a conglomeration of dentin and osteocementum. A portion can be recognized as crown. Beneath the crown is a cavity containing bacterial colonies, detritus, and exudate. Areas which are partly formed and partly resorbed are filled with soft tissue which cannot be identified definitely as pulp. These areas are surrounded by osteocementum with islands of dentin. The portion identified as root shows a condensed pulp with few denticles. The wall shows a seam of dentinoid and the apical portion is irregular, being partly made up by osteocementum. The soft tissue throughout shows infiltration by lymphocytes and plasma cells. This is moderate except where the gingival attachment would be and here the surface is covered with a squamous epithelium. The section of the soft tissue taken from here is covered with a broad lining of squamous epithelium with marked subepithelial infiltration by plasma cells and lymphocytes.

The soft tissue received separately shows similar epithelial lining resting upon connective tissue. The opposite edges show epithelial covering as seen in the oral cavity. There is slight parakeratosis. Another particle considered as cyst wall shows at the periphery remnants of bone with osteocytes in lacunae.

Diagnosis: The first molar shows viable pulp with moderate focal chronic inflammation in the pulp chamber. There are pulp denticles and hypercementosis of the roots.

The malshaped supernumerary tooth shows a conglomeration of dentin and osteocementum. The pulp is free of inflammatory reaction. The soft tissue filling the external resorbed areas shows chronic inflammatory reaction.

The cyst wall surrounding the coronal portion of the supernumerary tooth and that received separately shows marked chronic inflammatory reaction—comparable with follicular cyst.

M. Freund, M.D.

COMMENT

The problem arising in this situation was not one of treatment of a cystic lesion involving a supernumerary tooth but, rather, one of diagnosis to determine actual or possible antral involvement by the lesion. Since an infectious process was present at the initial examination no Lipoidol or other opaque medium was used to visualize the extent of the growth because of the possibility of inflaming an otherwise quiescent area. Direct probing was also impossible because of the thickened nature of the lesion. Instead, multiple roentgenographic studies were employed to determine the extent of the lesion as well as the possible antral involvement.

The Waters' view film of the antra is the most important roentgenographic film that can be obtained in this situation, as both antra can be visualized and compared simultaneously. It is of invaluable aid in determining the superior and lateromedial movements of the cystic expanse from its inception in the molar area. In the case just presented it was noted through this means that the right antrum was displaced superomedially and not infiltrated by the expanding sac. This materially lessened the danger of the cyst removal and aided in the patient's rapid recovery from the surgical procedure.

SUMMARY

1. A case of a dentigerous cyst involving a supernumerary maxillary molar with encroachment on the maxillary antrum is reported.

2. The problem of diagnosing antral involvement in this situation is presented and discussed briefly.

3. Multiple roentgenographic views of this area, including Waters' views and lateral projections of the antra, are recommended as excellent diagnostic aids.

4. It is suggested that encroachment on the maxillary antra by cystic lesions may be a more common finding than heretofore suspected.

EXODONTIA

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FRACTURES OF THE MAXILLARY TUBEROSITY OCCURRING DURING TOOTH EXTRACTION

Lawrence Cohen, B.Ch.D., F.D.S.R.C.S., L.R.C.P., M.R.C.S., London, England

OCCASIONALLY during the course of extraction of the maxillary second or third molar the maxillary tuberosity may be fractured and can be felt to be moving with the forceps. The operator is then confronted with the problem of whether or not to proceed with the extraction. If he does proceed, he must be prepared to dissect out the fractured tuberosity; in so doing, he is faced with a large oroantral communication. The predisposing causes of a fractured maxillary tuberosity are a large maxillary sinus with thin walls and a tooth with large divergent roots or an abnormal number of roots. A chronic apical infection of the affected tooth may result in bone sclerosis and render the bone of the tuberosity more liable to fracture.

If a tooth is being extracted because of severe pain, it cannot be left in situ. The only course available is to remove the molar together with the attached tuberosity. When the maxillary tuberosity bears only one tooth, a vertical incision is made from the buccal sulcus to the mesial surface of the affected tooth and is continued around the gingival margin of the tooth. When two teeth are involved, the vertical incision is made from the buccal sulcus to the mesial surface of the anterior tooth and is continued around the gingival margins of the two teeth. With careful dissection, the fractured tuberosity is removed. In order to avoid introducing infection, no attempt is made to aspirate blood clot from the maxillary sinus. A large defect is left which is closed by careful suturing with black silk. The use of one or two horizontal mattress sutures produces better apposition of the margins, and the remainder of the defect is closed with interrupted sutures. The patient is given a five-day course of intramuscular penicillin to prevent infection of the maxillary sinus on the affected side. An acrylic dental plate may be constructed to support the wound margins, but healing will occur without it. The patient is warned not to rinse his mouth forcibly or to blow his nose for

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at least a fortnight. There is usually slight bleeding from the nostril on the affected side for a day or two, and the patient should be warned of this.

It is important that the patient be treated in consultation with an ear, nose, and throat surgeon, who will deal with any possible infection of the affected sinus. The sutures may be removed on the fourteenth day, and it is advisable that follow-up roentgenograms of the affected sinus be taken after two months to ascertain whether healing has occurred satisfactorily.

If the maxillary tuberosity is fractured during the course of a dental clearance and the affected tooth has caused no pain, the tooth may be retained for two months and then removed surgically. Its antagonist in the mandible may be extracted to prevent pain on occluding the teeth, or the tooth may be splinted by means of a cap splint or arch wire. The patient is given a five-day course of intraumuscular penicillin to prevent infection.

CASE HISTORIES

CASE 1.—A 25-year-old man presented for extraction of painful upper right second and third molars under nitrous oxide-oxygen anesthesia. While the third molar was being extracted, the whole of the tuberosity was felt to move. The anesthetic was stopped and, on recovery, the region of the two teeth was infiltrated with 2 per cent Xylocaine.

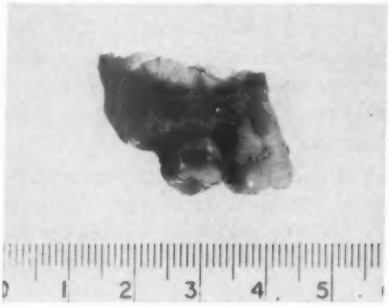

Fig. 1.

An incision was made on the buccal aspect of the upper right second molar, commencing in the buccal sulcus and extending to the mesial aspect of this tooth. The incision was continued around the gingival margins of the second and third molars. A buccal mucoperiosteal flap was raised, and the detached tuberosity was carefully dissected out. The incision was closed with interrupted sutures, and penicillin was given intramuscularly for five days. After fourteen days the sutures were removed. The patient was seen by an ear, nose, and throat surgeon for follow-up examination of the affected sinus. Healing was uneventful. Fig. 1 shows the tuberosity after removal.

CASE 2.—A 29-year-old man presented for extraction of a painful upper right third molar under local anesthesia. The tooth was found to be very firm, and during its removal the tuberosity was felt to be mobile. A buccal mucoperiosteal flap was raised, and the fractured tuberosity was dissected out. The incision was closed with interrupted sutures, and penicillin was given intramuscularly for five days. Healing was uneventful, and the ear,

nose, and throat surgeon reported a normal sinus on that side when the follow-up roentgenogram was taken. Fig. 2 shows the specimen after removal. Roentgenographic examination of the specimen revealed that this upper third molar had five roots.

Fig. 2.

DISCUSSION

The removal of a tuberosity will increase the difficulty of fitting a denture at some future date, but this is not an insurmountable problem. The conservative treatment of a fractured tuberosity with surgical removal of the affected tooth after two months will not markedly affect the shape of the alveolus and will give better retention of a denture. In the two cases presented here removal of the tuberosity was undertaken, since the patients attended because of pain in the maxillary molars.

SUMMARY

The treatment of fractures of the maxillary tuberosity occurring during tooth extraction is discussed and two cases are described.

I should like to thank Mr. D. Greer Walker for his advice and criticism in the preparation of this paper.

ORAL MEDICINE

COMPARISONS OF HISTOLOGIC, CYTOLOGIC, AND CLINICAL FINDINGS IN INTRAORAL LEUKOPLAKIA AND ASSOCIATED CARCINOMA

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LEUKOPLAKIA or white patch is a lesion which often occurs on the mucous membrane of the oral cavity. Occasionally carcinoma develops in association with this lesion, suggesting that the white patch may be a precursor to malignancy. The poor prognosis which accompanies these rather infrequent associations justifiably arouses concern. Furthermore, frequent inconsistencies between the clinical and microscopic findings many times confuse the diagnosis and complicate the management.

The purpose of this article is to present several case histories of patients with intraoral leukoplakia to demonstrate cytologic and histologic findings that are inconsistent with the clinical manifestations. In addition, the use of exfoliative cytology as an adjunct to biopsy to help the clinician evaluate these inconsistencies will be discussed.

REVIEW OF PERTINENT HISTORY

Establishment of a cause-and-effect relationship would simplify management in most cases. However, the cause of intraoral keratoses is undoubtedly manyfold and often not apparent. Smoking and exposure to coal-tar products have long been under suspicion.[2] Yet, clinical evidence indicates a more basic second or predisposing factor. This is evidenced by the unpredictable manner in which patients manifest the white patch. Cooke[3] indicated that the most common form of intraoral white patch arises from frictional irritation. Several authors[3, 6, 7, 12, 17] have classified keratotic lesions of the tongue by implying that

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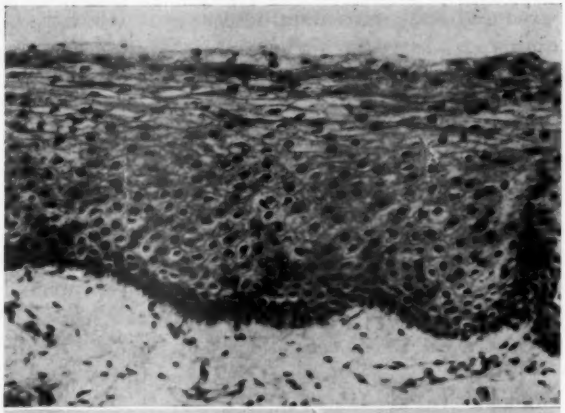


Fig. 1.

Fig. 2.

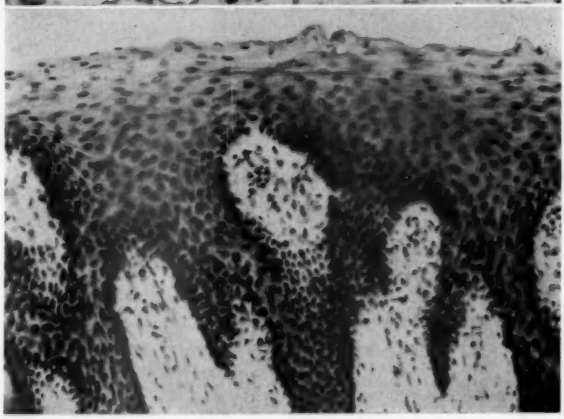


Fig. 3.

Fig. 1.—Normal human soft palate. (Magnification, ×235; reduced ¼.)
Fig. 2.—Normal human buccal mucosa. (Magnification, ×235; reduced ¼.)
Fig. 3.—Normal human gingiva. (Magnification, ×235; reduced ¼.)

cancer in situ produced a typical clinical appearance. However, the literature is scanty concerning any correlative study whereby clinical appearance would provide a reliable basis for prediction. Observations during our study indicate a definite lack of correlation and emphasize the danger of depending on clinical appearance alone. Many investigators[2, 5-7] agree that dyskeratosis is the deviation from normal which marks the lesion as dangerous. This alteration can be determined only microscopically. Since premalignant changes cannot be determined clinically,[2, 5, 6] it has been suggested that all persistent white patches or those showing signs of ulceration or induration should be biopsied.

Until recently, the value of exfoliative cytology in conjunction with leukoplakic lesions was questionable. Reports revealed nothing except highly cornified material characteristic of superficial epithelial cells.[8, 18] More recent investigations, however, have suggested the value of this technique in cases of leukoplakia associated with carcinoma in situ.[4, 9, 10, 16]

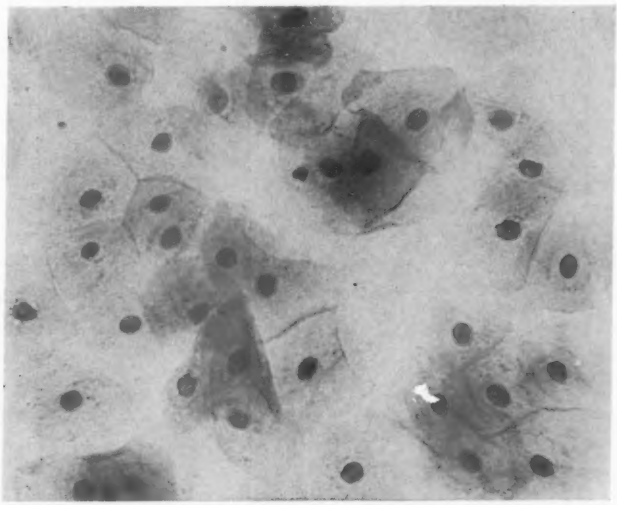


Fig. 4.—Squamous cells smeared from clinically normal buccal mucosa. There is uniformity in size and shape of the nuclei, which also display even chromatin distribution. Surrounding cytoplasm is uniform and abundant (small nuclear-cytoplasmic ratio). Staining reaction of the cytoplasm was approximately half acidophilic and half basophilic.

NORMAL HISTOLOGIC VARIATIONS

Variations of normal intraoral histology will be reviewed, since it appears that the character and thickness of the epidermal structure account for the change in the mucosa from normal epithelial translucency to whiteness with all its assorted features. Even normal oral mucosae show recognizable histologic variations in thickness and cornification (Figs. 1, 2, and 3). These features are mainly attributed to their particular anatomic location and function.

Nevertheless, the configuration of the epithelial ridges, the thickness of the prickle-cell layer, and the cornification of the stratum corneum are nearly constant for each representative area. Regardless of these anatomic differences, the various regions of mucosa present a rather translucent, pinkish appearance. Elongation of the epithelial ridges and an increase in thickness of the prickle layer (acanthosis) and the stratum corneum (hyperkeratosis) are the most consistent histologic features of oral keratoses.[12]

CYTOLOGY

Cells shed from the mucosa of the mouth also reflect differences which are manifested in cytoplasmic staining reactions.[18] Cells from areas of the mouth subject to functional trauma, such as the hard palate, gingiva, and tongue, show extensive cornification as evidenced by the relative acidophilia, while cells from the soft palate, buccal and labial mucosae, and floor of the mouth show a greater basophilic response. The nuclei and cytoplasm maintain a relatively constant size and shape (Fig. 4). As cornification and hyperkeratosis progress, cytoplasmic acidophilia and the incidence of denucleated cells increase.

CASE REPORTS

CASE 1.—A 47-year-old white man sought treatment because of a diffuse white area on the right buccal mucosa of three months' duration with apparently rapid spread (Fig. 5). The patient's history failed to disclose any obvious irritative factors. The lesion was firm

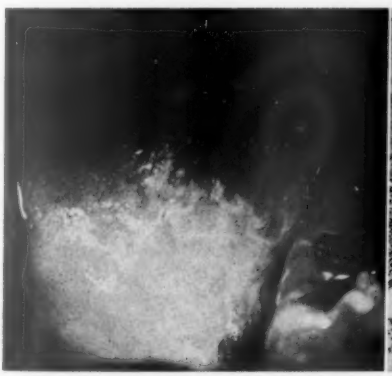


Fig. 5.

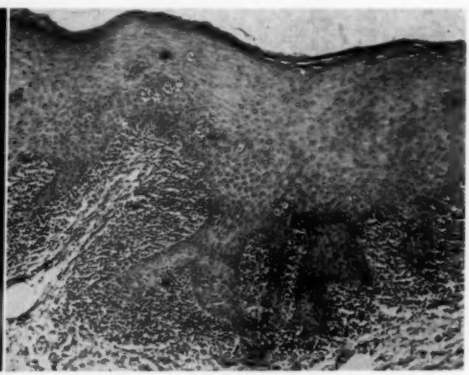


Fig. 6.

to palpation and slightly fissured. Minimal circumscribed inflammatory changes were observed. Because the history and clinical findings were nonspecific, an incisional biopsy was performed; this revealed changes which appeared compatible with lichen planus. No evidence of malignant or premalignant change was observed in the tissue sections (Fig. 6). Following healing of the wound, the site resumed its previous clinical appearance. After one year of observation, there has not been any significant clinical change except for occasional burning sensations in this area. Future diagnostic procedures and therapy will depend upon progression of signs and symptoms. Certainly, extensions, induration, and ulceration would

Fig. 7.

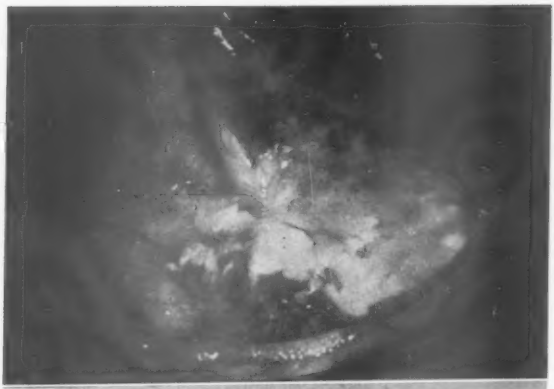


Fig. 8.

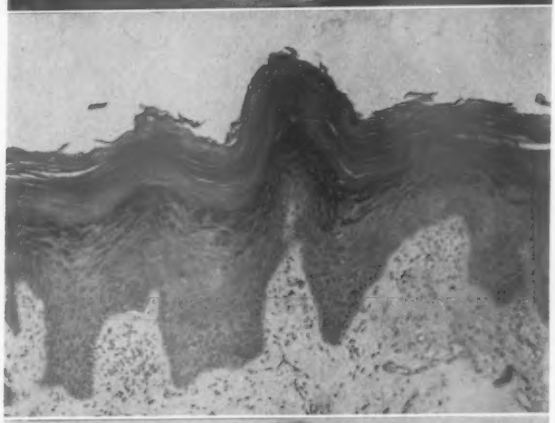
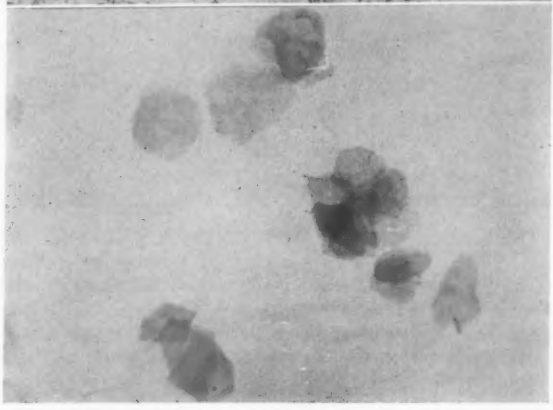


Fig. 9.



prompt further tissue study. Nonspecific keratotic lesions of the lichen planus type may present alarming clinical appearances which preclude adequate evaluation without microscopic tissue examination.

CASE 2.—A 73-year-old diabetic woman was referred by her physician with a tentative diagnosis of leukoplakia on the floor of the mouth. The patient had been aware of the lesion for five months before seeking treatment (Fig. 7). Because of the widespread, heaped-up appearance of the lesion, three representative biopsy specimens were secured. A mild acanthosis, an increase in the granular-cell layer, and a marked hyperkeratosis without the usual parakeratosis for this area were the only significant microscopic findings in the epidermis (Fig. 8). The cells of the epidermis showed orderly nuclear maturation. A slight inflammatory response was evident in the dermis beneath the white patch. Conservative removal of the lesion in its entirety was prompted by the bizarre clinical appearance. Histologic premalignant changes could not be demonstrated by serial histologic sections. This case illustrates the lack of correlation between a seemingly advanced and dangerous leukoplakic area and the orderly tissue pattern responsible for this clinical change. Prognosis of cancerous proliferation in this tissue would be only on the basis of speculation.

Exfoliated cells (Fig. 9) demonstrated an increase in cytoplasmic cornification, washed-out staining properties, and associated denucleation. This represents a typical cytologic finding from a nonulcerated hyperkeratotic area where only superficial, highly cornified cells are being obtained.

CASE 3.—A 42-year-old white woman was first seen for evaluation of a small, soft, lacy white patch on the floor of the mouth overlying the left plica sublingualis (Fig. 10). Because of the innocent appearance of this white patch, the patient was told to cut down on cigarette smoking and to return for further examination in three months. A checkup six months later showed no further change except for a slight inflammatory reaction at the periphery of the lesion; the patient complained of occasional burning in the floor of the mouth. The consultants felt that a further decrease in smoking, accompanied by palliative care, was adequate at this time. A routine cytologic smear from the lesion (Fig. 11) revealed epithelial cells consistent with malignancy. An incisional biopsy was prompted by the cytologic findings. The histologic analysis revealed multiple carcinomas in situ, with one area representing early invasion of the underlying connective tissue (Fig. 12). The patient was referred for a more radical excision because of the lesion's questionable borders. This case illustrates an instance in which clinical judgment and experience proved to be insufficient. A more conservative program of observation and its concomitant delay would have dimmed the prognosis for this patient. Without the diagnostic adjunct of cytologic studies, this patient might have become a hopeless therapeutic problem.

CASE 4.—A 70-year-old white man was being followed in the tumor clinic because of intraoral leukoplakia of at least five years' duration. The heaviest plaque was diffusely spread over the lower left alveolar mucosa (Fig. 13). The lesion remained unchanged in response to conservative methods of treatment which consisted of the patient's not wearing his dentures, cessation of smoking, and a restricted diet of soft, nonspicy foods. The patient was referred to the dental medicine clinic for evaluation. A biopsy revealed marked elongation of the epidermal ridges, a severe inflammatory response, and some abnormal mitotic activity (Fig. 14). Because of the microscopic findings of pseudoepitheliomatous hyperplasia and questionable pathogenesis,[1, 18] excision of the entire lesion was urged. The patient was referred back to the tumor clinic with this recommendation. Because the lesion was so diffuse and apparently inactive, the Board members agreed upon a regime of close observation. There was no improvement during the next eight months, and removal was again recommended. A local wide excision was performed; this included a portion of the floor of the mouth and the mucosa overlying the posterior alveolar ridge. A tissue diagnosis of well-differentiated squamous-cell carcinoma was made.

CASE 5.—A 45-year-old man had a firm, slightly fixed and fissured, raised white patch in the lower labial vestibular fornix of the oral cavity (Fig. 15). He stated that he had

Fig. 10.

Fig. 11.

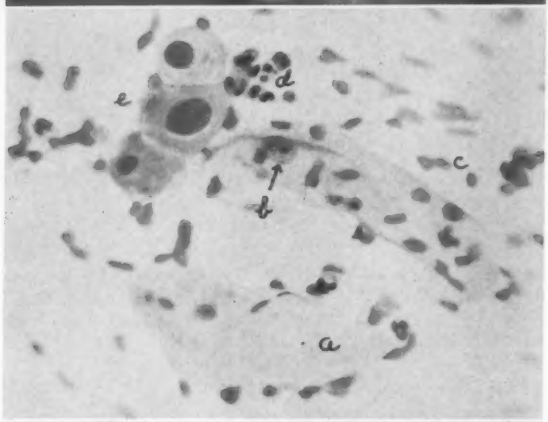


Fig. 12.

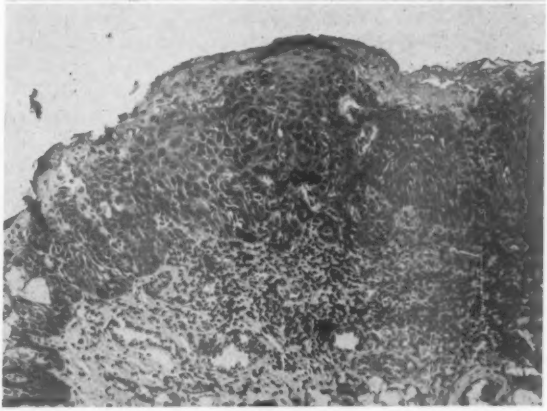


Fig. 10.—Arrows indicate lacy white patch on floor of mouth.

Fig. 11.—*A*, Normal cornified squamous cytoplasm without nucleus; *B*, normal squamous nucleus (approximately 12 microns); *C*, red blood cells (approximately 7.5 microns); *D*, polymorphonuclear leukocytes; *E*, three malignant squamous cells demonstrating nuclear pleomorphism, massive enlargement, hyperchromatism, and altered nuclear-cytoplasmic ratio.

Fig. 12.—Biopsy specimen shows carcinoma invading underlying connective tissue.

Fig. 13.

Fig. 14.

chewed tobacco for many years and frequently held it in this region. The patient was told to abstain from chewing until further evaluation of his lesion was accomplished. Squamous-cell carcinoma was strongly suspected. A histologic specimen obtained by a punch biopsy showed acanthosis and hyperkeratosis with only a slight evidence of inflammation (Fig. 16). The lesion completely disappeared in twelve days. The patient now smokes cigarettes, and observations over a three-year period have shown no further keratotic activity. Elimination of the specific irritant will often reverse the keratotic process. However, clinical criteria for predicting response or cellular threshold of irritant tolerance remain an enigma.

Fig. 15.

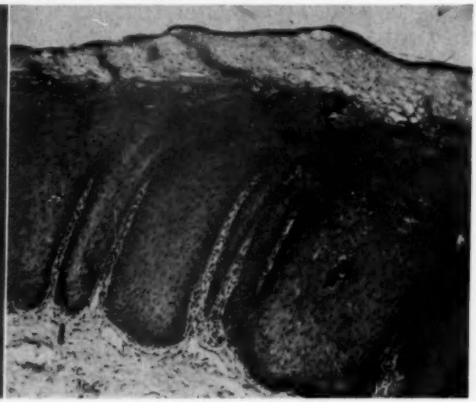


Fig. 16.

Case 6.—A 48-year-old white man, who was a heavy cigarette smoker, noticed a white patch (Fig. 17) on the floor of his mouth. A biopsy of a questionable area of the lesion (Fig. 17, *A*) by a local physician showed no malignant activity. The patient was referred for cytologic examination, since the lesion would not heal and carcinoma was suspected. A smear of the lesion (Fig. 18) displayed the cellular features of clumping, pleomorphism, hyperchromatism, enlarged nuclei, and abnormal clearing. Because of the observed malignant

Fig. 17.

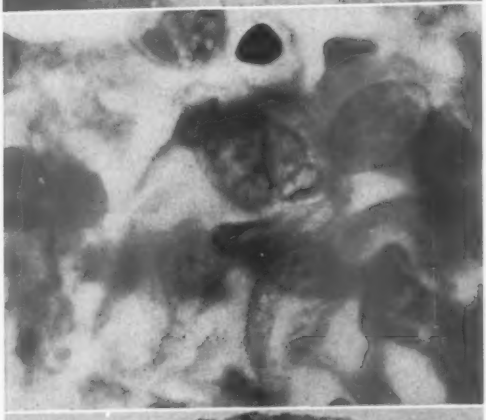


Fig. 18.

Fig. 19.

cells, wide excision was advised. The surgical specimen (Fig. 19) revealed squamous-cell carcinoma. This case illustrates that a single incisional biopsy may not be representative or adequate to rule out malignant activity.[1] In the event of continued clinical suspicions, further diagnostic procedures and close observation are indicated.

DISCUSSION

The preceding cases illustrate the extreme variation which may occur between the clinically observed intraoral white patch and the histologic appearance of the tissue. Prediction of a histologic pattern has been unsatisfactory. Indications for biopsy have been shown to be ill-defined. Furthermore, false negative biopsies are sometimes obtained which confuse the diagnosis. These false tissue diagnoses occur because only a small portion of the entire lesion has undergone malignant change at the time the biopsy was performed. Such misleading interpretations should encourage more extensive tissue sampling, serial histologic sections, and careful postbiopsy observations. Exfoliative cytologic examination is a useful adjunct in these instances. In two cases presented here, positive cytologic smears prompted biopsy of early malignant lesions which, due to lack of clinical signs, probably would not have been biopsied at that time. The attributes of this procedure are derived from their accuracy, speed, lack of pain, and simplicity.[18] This technique permits immediate cellular evaluation, extensive sampling of the lesion, and uncomplicated follow-up studies. While false positives have not been experienced with this method, false negatives may occur as they do in the biopsy.

If it were not for the occasional association with carcinoma, diagnosis and management of the intraoral keratotic lesion would be of little concern. The early diagnosis of potentially serious lesions is especially desirable because of the much better prognosis that is associated with early treatment.

SUMMARY

Histologic changes, such as acanthosis, hyperkeratosis, and dyskeratosis, may all be responsible for the intraoral white lesion. In many instances, however, correlation is lacking between the clinical appearance and the histologic pattern. Six cases are presented to illustrate this fact. An innocent-appearing white patch may represent an early carcinoma. Proper diagnosis and management of these lesions may be made possible by rational use of cytologic smears, biopsy techniques, and careful observation of response to therapy.

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PHARMACOLOGY AND THERAPEUTICS

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DENTAL USE OF MEPROBAMATE AND PROMAZINE HYDROCHLORIDE FOR MENTALLY RETARDED AND EMOTIONALLY DISTURBED PATIENTS

A Preliminary Report

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DENTAL treatment of the mentally handicapped and those who have severe emotional difficulties sometimes presents a problem requiring the use of a general anesthetic before the required procedures can be performed. The degree of disturbance present may range from simple emotional disorders to the mental retardation of the moron, idiot, and imbecile. The goal in management of such patients is to maintain these persons in a cooperative state so that they will submit to the necessary treatment and yet not to depress them to such an extent as to interfere with ambulation. In earlier clinical experience some of the ataraxics, used alone, had been found valueless in preparing patients for painful dental manipulations. The apprehension of the patient overshadowed and counterbalanced the degree of relaxation obtainable with a single ataractic agent.

Pharmacotropic drugs function in various ways. Meprobamate, for example, acts principally on the interneuronal pathways of the thalamus and spinal cord and lessens emotional reactivity.[1] The electroencephalographic tracing in animals shows a pronounced decrease in thalamic activity.[2] Promazine hydrochloride, a phenothiazine derivative, acts primarily on the reticular-activating system of the hypothalamus.[3] Promazine hydrochloride induces a definite taming effect in monkeys and controls the motor hyperactivity produced by morphine in cats.[4] In clinical use, meprobamate has a sedative and muscle-relaxing effect. Promazine hydrochloride tends to calm mental disturbances and subdue motor overactivity.

For control of the exaggerated forms of anxiety, tension, and apprehension, we were interested in obtaining an agent which would produce emotional relaxation as well as suppress tendencies to abnormal behavior. In view of the experimental and clinical reports concerning the action of meprobamate

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and of promazine hydrochloride, it was considered desirable to use a combination of both,* in capsule form, in the hope of enhancing, with a minimum of side reactions, the differing pharmacologic properties of each.

PLAN OF STUDY

A group of twenty-five patients, fourteen of whom were homebound and eleven of whom had a background of institutional care, were selected for a course of medication with the capsules before dental treatment. All were emotionally disturbed and mentally retarded and had been difficult to handle in previous clinical experience, but all were completely harmless. Their behavior under medication, therefore, could be compared to what their response had been to treatment under so-called "normal" circumstances.

No young children were included in the group; the patients' ages ranged from 8 to 35 years. All were able to enter the dental clinic of their own volition and, in most cases, to seat themselves in the dental chair.

The institutionalized group ranged in intelligence from high-grade morons to low-grade imbeciles. Previous medication for these patients consisted of Dilantin sodium, phenobarbital, and chlorpromazine hydrochloride.

The fourteen homebound children were emotionally disturbed and represented the classic athletic type of true mental retardation.

MEDICATION

The medicated and placebo capsules were prepared and coded. To eliminate bias on the part of the investigators, the contents of the capsules were not known to the participating dentists.

For best results, it was felt that the medication should be continued for at least four days before the time of actual dental treatment and that a maximum of one week might be desirable to create a "build-up" period. When medication was administered on the day of the dental procedure, it was given at least one to two hours prior to the patient's visit to the clinic.

The treatment program was carried out in several phases. The first group to receive medication, the four most severely retarded institutionalized patients, were treated with one capsule three times a day for four days. The patients in the second group received one capsule four times a day for two days, then two capsules three times a day for two days, and finally one capsule three times a day for one week. In subsequent patients the dose was initiated at one capsule four times a day for two days and two capsules three times a day for two days. The placebo was administered in the same dosage in the week preceding the next appointment.

Nine of the most severely retarded homebound patients received one capsule three times a day during the week before scheduled dental treatment, and the placebo was administered in the same dosage in the week prior to the next visit. Five of the group (children) who were less emotionally disturbed

*Prozine (200 mg. meprobamate, 25 mg. promazine hydrochloride), a product of Wyeth Laboratories, Philadelphia, Pennsylvania.

received one capsule four times a day for two days and then two capsules three times a day for two days. In the placebo period, preceding the next appointment, these patients received meprobamate alone for comparison. The dosage was the same as for the capsules containing the combination (200 mg. per capsule).

Supplementary medication with anticonvulsants or other agents was continued without interruption.

RESULTS

Best results in relaxation and cooperation were obtained when the emotional disturbance was at a minimum. Some improvement in behavior during treatment occurred in 52 per cent of the total series. When the placebo was administered, all patients reacted as though no medication had been given.

Only one of the four most severely retarded of the institutionalized group showed definite relaxation and cooperation after having received the medicated capsules. This patient, for whom treatment previously had been impossible because of her severe apprehension, actually allowed dental procedures to be carried out and roentgenograms to be taken of her teeth. Two others of the four permitted the dentist to clean their teeth, and one for the first time allowed herself to be seated in the dental chair but would not submit to dental work.

The remaining seven institutionalized patients, while awaiting dental treatment, were less apprehensive and more relaxed after having received the medication than after taking the placebo. All eleven of the institutionalized group were less boisterous in the waiting room after the medication.

The five less emotionally disturbed of the homebound patients reacted favorably to the medication and permitted dental treatment, whereas after administration of the placebo there was much apprehension and movement. If relaxation and cooperation occurred in some degree after administration of meprobamate alone, the response was not to be compared with the results obtained with the combination.

The nine most severely afflicted of the homebound group showed no response to either the medicated capsules or the placebo. Meperidine, 50 mg., was administered intramuscularly one hour prior to the dental appointment, in addition to the prescribed dose of the meprobamate-promazine hydrochloride combination. Four patients reacted somewhat more favorably to these measures and appeared more cooperative. Although there was some movement, some dental work was accomplished. For the remaining five (20 per cent of the series), it was necessary to administer a general anesthetic in order to carry out treatment.

When the response to medication shown by the institutionalized group was compared with that of the homebound patients, it was seen that those who lived in an atmosphere completely removed from parental influences seemed more cooperative and willing than those who lived in a home environment.

The patients who were rendered more cooperative by the medication appeared to have some lessening of salivation. This could have been the result of their relaxation and the reduction of their anxiety and fear of dental treatment.

No adverse reactions developed.

SUMMARY AND CONCLUSIONS

A preliminary double-blind study of the relaxation during dental treatment produced by premedication with capsules containing 200 mg. meprobamate and 25 mg. promazine hydrochloride was carried out in twenty-five mentally retarded, emotionally disturbed patients. Administration of the medicated capsules was alternated with administration of placebos. Premedication was continued for a minimum of four days.

The severity of the mental deficiency influenced the success of the medication, regardless of the dose used. The less disturbed were more relaxed under dental treatment, and the institutionalized patients were more cooperative than the homebound. A favorable result was obtained in 52 per cent. In the severely retarded, little or no behavioral improvement occurred. After having received the medicated capsules, all the institutionalized patients were less boisterous in the office while awaiting treatment. Placebos were ineffective in all cases. Salivation was lessened in those patients who responded to premedication.

For the most severely retarded, who had failed to respond to the capsule medication, meperidine was added to the dosage, with favorable effects in some cases. Twenty per cent of the series required a general anesthetic.

Daily medication with anticonvulsants and other agents was not interrupted. No adverse reactions developed.

Although the results of this study were most encouraging, further work on a larger scale is necessary. The relaxation and cooperation obtained in different groups will vary. The desired effect does not appear to be obtainable on a single dosage level. Addition of an analgesic also may be required, and it may be advantageous to continue premedication over a longer period to enable the desired build-up of relaxation.

Appreciation is given to Dr. L. T. Longtin and his dental staff of the Elwyn Training School for their cooperation in this study.

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Medical Arts Bldg.

ANESTHESIOLOGY

CARDIAC ARREST

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SINCE the dental practitioner constantly makes use of both local and general anesthetics, it is important that he know something about cardiac arrest. This is the most serious complication that can occur during any procedure. Therefore, knowledge of the basic principles underlying its etiology, diagnosis, treatment, and prevention is important.

ETIOLOGY

Cardiac arrest may be defined as a standstill of heart action. Although the specific cause or causes may not be entirely obvious in some patients, a deficiency in the amount of oxygen to the vital tissues is the basic factor underlying most cardiac complications. Oxygen deficiency may be due to impaired ventilation, impaired circulation, or both.

Experiments have shown that cardiac muscle is able to support only a small oxygen debt, and normal function ceases when the debt amounts to 0.06 c.c. per gram of tissue. On the other hand, skeletal muscle is capable of contracting an oxygen debt up to 0.30 c.c. per gram of tissue. Cardiac muscle is very sensitive to changes in pH and has a low buffering power. Therefore, when lactic acid, which is formed during heart action, reaches a concentration of approximately 0.07 per cent in heart muscle, such irregularities as extrasystoles and heart block occur, followed by cessation of the heartbeat.[1]

Cardiac reflexes are hyperactive during myocardial hypoxia.[1] The resulting hypercapnia activates the vagal reflexes centrally. Situations leading to the above conditions may occur in response to the following:

1. Excess sedation causing respiratory depression.
2. Patient's position.
3. Inadequate airway.
4. Carrying the patient at too deep an anesthetic level, causing respiratory depression.

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5. Too little oxygen in the anesthetic mixture.[2]
6. Anemia.
7. Hypotension.
8. Abnormal respiratory function due to respiratory disease.
9. Reflex phenomena due to endopharyngeal, endotracheal, or endoesophageal manipulation or painful peripheral nerve stimuli may initiate cardio-inhibitory responses.[3]
10. Myocardial disease, causing intrinsic disturbances of rhythm, and myocarditis make for abnormal oxygen utilization.
11. Metabolic conditions affecting the myocardium, such as:
    (a) Myocardial damage due to renal failure, syphilis, and arteriosclerotic heart disease.
    (b) Hyperkalemia or hypokalemia.
12. Overdosage of anesthetic agents, causing myocardial or central nervous system toxicity.[3]
13. Use of cyclopropane or trichlorethylene with Adrenalin may cause arrhythmias.[3]

DIAGNOSIS

If the patient is to survive, prompt recognition of the condition and immediate emergency measures must be instituted. There may be no warning signs present. On the other hand, arrhythmias or bradycardia with a fall in blood pressure may occur. The only means of prompt diagnosis is constant monitoring of pulse and blood pressure.

If the pulse and blood pressure cannot be obtained, heart sounds are inaudible, and pupils are widely dilated, then a diagnosis of cardiac arrest is made. Time is the most important factor at this stage, for cortical damage is seldom reversible after more than four minutes of complete anoxia. The extent of severity of neurological complications is in direct proportion to the delay between arrest and massage, with adequate oxygenation of the cortical cells.[2]

RESUSCITATION

The emergency resuscitation procedure is accomplished by re-establishing the oxygen supply system within four minutes and restoring the heart beat.

The oxygen system is restored by getting oxygen directly into the lungs by the use of bag and mask. At the first opportunity, an endotracheal tube should be placed in position to ensure an adequate airway. The oxygenated blood must be delivered by cardiac massage to the brain and other vital organs within four minutes from the time of arrest.

In order that the necessary procedures for cardiac massage may be accomplished as quickly and as accurately as possible, thoracotomy is essential. Some of the advantages of a thoracotomy are as follows:

1. It is possible to visualize the heart directly, thus making sure of what it is doing.

2. The pericardial sac may be opened easier.
3. Direct cardiac massage results in greater cardiac output than cardiac massage performed through the abdomen.
4. Electrical defibrillation is easier.
5. The aorta may be clamped off to shunt the blood.
6. Drugs may be injected with greater accuracy.

Time should not be wasted by using a sterile procedure. A transverse incision is made in the left fifth interspace, and the ribs are spread apart. The heart is then grasped in the left hand with the thumb on the left side and the fingers on the right side of the heart. The rate of massage is between thirty-five and fifty times per minute. This usually gives adequate peripheral circulation and blood pressure. If a good peripheral pulse is maintained and the pupils are constricted, all efforts should be kept up.

RESTORATION OF HEARTBEAT

Cardiac standstill occurs more frequently than ventricular fibrillation. This is fortunate because cardiac standstill is the easier condition to treat.

In ventricular standstill, the heart may be contracting feebly or not at all. Massage should be performed for one or two minutes before stimulants are injected, for many cases respond to massage alone. Often ventricular fibrillation occurs from the injection itself or from the drug injected.

The left ventricle is the area of choice for the injection of drugs; the substance injected reaches the coronary and myocardial circulation in a shorter period of time than if another site is used.

Adrenalin is a cardiac stimulant. It augments and increases the heart rate. A quantity of 0.2 c.c. to 0.3 c.c. of a 1:1,000 solution of Adrenalin is used. A small dose is first given and repeated if necessary, rather than a large dose, which may lead to ventricular fibrillation.[4]

Calcium chloride increases myocardial excitability and contractility, prolonging systole.[5] As the serum calcium increases, there is vagal bradycardia. Also, there is a direct action on the ventricular muscle which causes increased excitability of ventricular rhythm, resulting in extrasystoles. The dose is 10 c.c. of a 5 per cent solution or 2 c.c. to 4 c.c. of a 10 per cent solution injected into the left ventricle. The drug is helpful when the heart is refractory to Adrenalin. Sometimes heparin, 1 mg. per cubic centimeter, is injected simultaneously into the ventricle to reduce the hazard of coagulation from an excessive amount of calcium.

Atropine, 0.6 mg., may be given intravenously. This blocks interference with stimulus formation in the auricles and diminishes the effect of vagovagal reflexes.[6]

VENTRICULAR FIBRILLATION

Ventricular fibrillation is the incoordinate contraction of individual muscle fibers. Massage alone does not stop ventricular fibrillation, but it does prevent it from deteriorating from the early fine, rapid type to the slow, coarse type.

Chemical defibrillation is not always successful and, if successful, it takes from six to ten minutes.

Potassium chloride may be used. The potassium ion promotes relaxation of the myocardium. A quantity of 2 c.c. to 5 c.c. of a 0.5 per cent solution is injected into the left ventricle.[1]

Procaine, 10 c.c. of a 2 per cent solution, or Pronestyl, 500 mg., may be given. Procaine, however, is seldom effective because of its depressant action. Also, cardiac arrest is more difficult to treat when procaine is used.[4]

In 1951, Kay[7] advised against the use of procaine, unless serial defibrillation had failed.

Then 10 c.c. of 1 per cent procaine and an electric shock are given. If this is not successful, then 1 c.c. of a 1:1,000 solution of epinephrine or 5 c.c. of a 1 per cent calcium chloride solution is injected into the left ventricle and an electric shock is administered again. If the rhythm is slow and faltering, the Zoll Pacemaker may be applied to the chest.

Electric defibrillation is the method of choice. This produces cardiac standstill by means of an electric shock.[8] Attempts are then made to initiate spontaneous beats as discussed previously. Even if the heart is under the influence of procaine or Adrenalin, electric defibrillation produces cardiac standstill. If it is thought that fibrillation has lasted four minutes or more, cardiac massage should be done before electric shock is given, thus permitting competent ventricular contractions for a few minutes. Electrodes are placed firmly on the anterior and posterior surfaces of the heart. The usual strength of the electric shock is 110 volts to 270 volts, 1.5 amperes, for 0.1 second. If the patient's muscles do not contract, the current is not strong enough.[9]

POSTOPERATIVE CARE OF PATIENT

The endotracheal tube is left in place until the patient is conscious and breathes completely on his own. Oxygen is supplied, with an adequate airway, at all times. Also a tracheotomy set should be available for emergency treatment. The patient should be digitalized intravenously. The vital signs are taken every five minutes until stable. Infection is controlled with antibiotics. The temperature is watched carefully; a rapid increase may mean cerebral damage. Intake and output are observed for signs of complications. The following complications may occur:

1. Cardiac arrhythmias.
2. Cardiac massage may lead to traumatic hemorrhagic pericarditis. Myocardial damage is directly proportional to the duration of massage. It is possible that excessive massage may lead to cardiac rupture. A pericardial reaction is always present after massage. There is some damage to the myocardium if massage lasts longer than fifteen minutes.
3. Acute cerebral edema, which is the usual cause of death after restoration of the circulatory system. This may not appear for several days.

4. Hydrothorax and pneumothorax.
5. Hemorrhage.
6. Wound infection.

CONCLUSION

Some of the basic factors underlying the etiology, treatment, complications, and prevention of cardiac arrest have been discussed.

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NATIONAL BANK BLDG.

FACTORS CONTRIBUTING TO DIRECT RADIATION REDUCTION IN DENTISTRY

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THREE investigation projects are herein described; all have practical implications and, in a sense, are related. *Project 1* was undertaken for the purpose of determining the amount of radiation reduction possible through the use of various practical thicknesses of aluminum filters at different voltages on a dental x-ray machine. *Project 2* was undertaken in an attempt to determine how much radiation passes through the lead backing of an intraoral film at different kilovoltages when 2.5 mm. of added aluminum filtration and 0.5 mm. of inherent aluminum filtration are used. *Project 3* was carried out in an effort to determine the extent to which radiation can be reduced by using ultraspeed intraoral films in comparison with intraoral film of medium speed.

PROJECT 1

Materials Used in Investigations.—For measuring the reduction in radiation, a Keleket charger, Model K-135, and two Keleket dosimeters were used. One of the dosimeters measured from 0 to 200 milliroentgens, and the other measured from 0 to 5 roentgens. Aluminum filters, each 0.5 mm. in thickness, were used. Two dental x-ray machines of different makes were utilized. Each machine had an inherent filtration of 0.5 mm. of aluminum. Each machine was collimated with lead, so that the diameter of the direct umbra measured 2 inches in diameter at the end of the cone or pointer. An 8-inch focal-film distance was used throughout the procedure.

Methods of Investigation.—All measurements of radiation were made by placing the charged dosimeter on the buccal side of the mandibular molar region. An exposure of one second was made. The dosimeter was read, the radiation

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was recorded, and the dosimeter was recharged to 0. This same procedure was followed on the lingual surface. The technique was repeated with a two-second exposure. These measurements of radiation were repeated, starting with inherent filtration only and adding 0.5 mm. of aluminum after each measurement until a total of 2.5 mm. of aluminum filter was added. Voltages ranged from 45 KVP to 70 KVP. A constant milliamperage of 10 was used. All measurements were made in air. The x-ray cone tip touched the specimen on the buccal surface when lingual readings were obtained and was in contact with the dosimeter on the buccal surface when buccal readings were taken (Fig. 1).

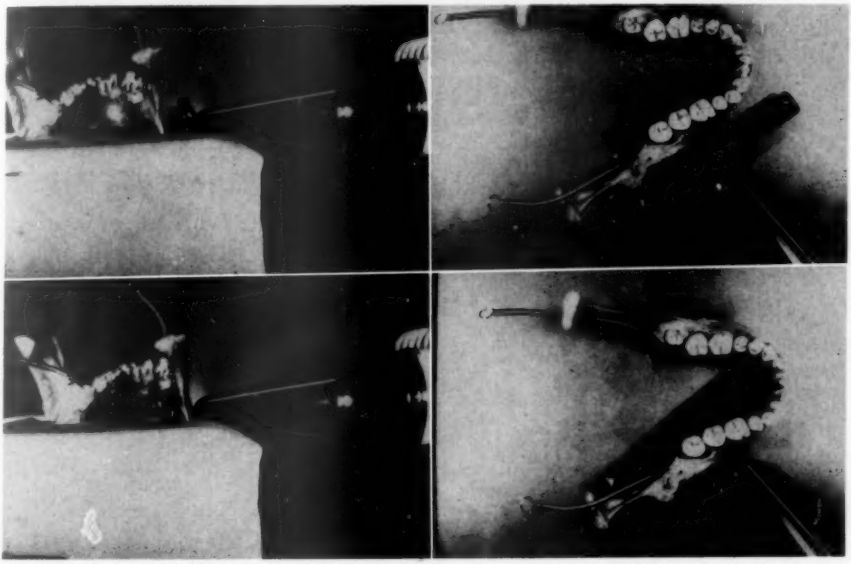


Fig. 1.

Results of Investigations.—Trout, Kelley, and Cathey[2] stated: "The radiation that is transmitted through the part is the useful radiation in making a roentgenogram. In the light of present understanding, the radiation that is not transmitted through the part can produce no useful effect." Table I shows the results obtained through the methods of Project 1.

Through the use of aluminum filtration, the buccal radiation was reduced 50 to 90 per cent; the lingual radiation was reduced only 10 to 30 per cent on the average.

As an example, at 65 KVP and 10 Ma., the radiation measured 1,500 mr on the buccal side when inherent filtration, at a one-second exposure was used. When 2.5 of aluminum filtration was added, this radiation was reduced to 400 mr, a 73.30 per cent reduction. Lingual radiation, using the same factors, was reduced from 70 mr to 60 mr, a reduction of 14.28 per cent.

TABLE I

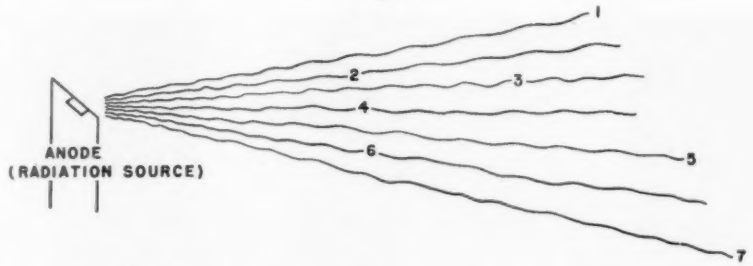
45 KVP — 10 Ma.	45 KVP — 10 Ma.	45 KVP — 10 Ma.	45 KVP — 10 Ma.	45 KVP — 10 Ma.	45 KVP — 10 Ma.
Inherent filter	0.5 mm. aluminum	1 mm. aluminum	1.5 mm. aluminum	2 mm. aluminum	2.5 mm. aluminum
1 sec. B 500 mr	1 sec. B 200 mr	1 sec. B 112 mr	1 sec. B 81 mr	1 sec. B 55 mr	1 sec. B 31 mr
1 sec. L 8 mr	1 sec. L 8 mr	1 sec. L 6 mr	1 sec. L 4 mr	1 sec. L 2 mr	1 sec. L 5 mr
2 sec. B 1,000 mr	2 sec. B 750 mr	2 sec. B 400 mr	2 sec. B 123 mr	2 sec. B 150 mr	2 sec. B 105 mr
2 sec. L 16 mr	2 sec. L 15 mr	2 sec. B 12 mr	2 sec. L 7 mr	2 sec. L 8 mr	2 sec. L 7 mr
50 KVP — 10 Ma.	**50 KVP — 10 Ma.**	**50 KVP — 10 Ma.**	**50 KVP — 10 Ma.**	**50 KVP — 10 Ma.**	**50 KVP — 10 Ma.**
1 sec. B 750 mr	1 sec. B 500 mr	1 sec. B 400 mr	1 sec. B 118 mr	1 sec. B 89 mr	1 sec. B 98 mr
1 sec. L 19 mr	1 sec. L 17 mr	1 sec. L 19 mr	1 sec. L 12 mr	1 sec. L 14 mr	1 sec. L 10 mr
2 sec. B 1,600 mr	2 sec. B 1,000 mr	2 sec. B 800 mr	2 sec. B 400 mr	2 sec. B 400 mr	2 sec. B 192 mr
2 sec. L 30 mr	2 sec. L 45 mr	2 sec. L 24 mr	2 sec. L 28 mr	2 sec. L 30 mr	2 sec. L 27 mr
55 KVP — 10 Ma.	**55 KVP — 10 Ma.**	**55 KVP — 10 Ma.**	**55 KVP — 10 Ma.**	**55 KVP — 10 Ma.**	**55 KVP — 10 Ma.**
1 sec. B 1,000 mr	1 sec. B 750 mr	1 sec. B 500 mr	1 sec. B 350 mr	1 sec. B 300 mr	1 sec. B 128 mr
1 sec. L 25 mr	1 sec. L 30 mr	1 sec. L 21 mr	1 sec. L 25 mr	1 sec. L 20 mr	1 sec. L 17 mr
2 sec. B 2,000 mr	2 sec. B 1,500 mr	2 sec. B 1,000 mr	2 sec. B 750 mr	2 sec. B 500 mr	2 sec. B 500 mr
2 sec L 61 mr	2 sec. L 80 mr	2 sec. L 69 mr	2 sec. L 50 mr	2 sec. L 50 mr	2 sec. L 40 mr
60 KVP — 10 Ma.	**60 KVP — 10 Ma.**	**60 KVP — 10 Ma.**	**60 KVP — 10 Ma.**	**60 KVP — 10 Ma.**	**60 KVP — 10 Ma.**
1 sec. B 1,500 mr	1 sec. B 1,000 mr	1 sec. B 600 mr	1 sec. B 400 mr	1 sec. B 350 mr	1 sec. B 350 mr
1 sec. L 51 mr	1 sec. L 62 mr	1 sec. L 46 mr	1 sec. L 32 mr	1 sec. L 31 mr	1 sec. L 39 mr
2 sec. B 2,500 mr	2 sec. B 1,750 mr	2 sec. B 1,500 mr	2 sec. B 1,000 mr	2 sec. B 800 mr	2 sec. B 600 mr
2 sec. L 100 mr	2 sec. L 149 mr	2 sec. L 110 mr	2 sec. L 90 mr	2 sec. L 72 mr	2 sec. L 64 mr
65 KVP — 10 Ma.	**65 KVP — 10 Ma.**	**65 KVP — 10 Ma.**	**65 KVP — 10 Ma.**	**65 KVP — 10 Ma.**	**65 KVP — 10 Ma.**
1 sec. B 1,500 mr	1 sec. B 1,000 mr	1 sec. B 600 mr	1 sec. B 500 mr	1 sec. B 400 mr	1 sec. B 400 mr
1 sec. L 70 mr	1 sec. L 88 mr	1 sec. L 71 mr	1 sec. L 61 mr	1 sec. L 67 mr	1 sec. L 60 mr
2 sec. B 2,750 mr	2 sec. B 2,000 mr	2 sec. B 1,600 mr	2 sec. B 1,300 mr	2 sec. B 1,100 mr	2 sec. B 900 mr
2 sec. L 150 mr	2 sec. L 210 mr	2 sec. L 168 mr	2 sec. L 139 mr	2 sec. L 159 mr	2 sec. L 140 mr
70 KVP — 10 Ma.	**70 KVP — 10 Ma.**	**70 KVP — 10 Ma.**	**70 KVP — 10 Ma.**	**70 KVP — 10 Ma.**	**70 KVP — 10 Ma.**
1 sec. B 1,500 mr	1 sec. B 1,100 mr	1 sec. B 750 mr	1 sec. B 650 mr	1 sec. B 700 mr	1 sec. B 500 mr
1 sec. L 110 mr	1 sec. L 110 mr	1 sec. L 105 mr	1 sec. L 100 mr	1 sec. L 90 mr	1 sec. L 95 mr
2 sec. B 3,000 mr	2 sec. B 2,500 mr	2 sec. B 2,000 mr	2 sec. B 1,650 mr	2 sec. B 1,250 mr	2 sec. B 1,200 mr
2 sec. L 240 mr	2 sec. L 265 mr	2 sec. L 400 mr	2 sec. L 300 mr	2 sec. L 181 mr	2 sec. L 195 mr

Machine—B.
Timer—Mechanical.
1,000 mr = 1 roentgen unit.
B = Buccal radiation.
L = Lingual radiation.

The accuracy and consistency of the recorded reading may be questioned. However, the trend of these numerous readings establishes a basic concept, namely, that increased aluminum filtration reduced radiation to both the buccal and the lingual areas but the reduction is far greater on the buccal side than on the lingual side.

A further explanation of the results is indicated. The greater the thickness of aluminum filtration, the greater the reduction of the long wave length radiation included in the direct beam. For example, let us assume that a beam of radiation is represented by seven different wave lengths. Fig. 2 illustrates seven x-rays or photons, all of which have varying wave lengths. Because there is no intervening structure, each ray is able to travel approximately the same distance. Quite arbitrarily, rays 1, 3, 5, and 7 are designated as having long wave lengths and rays 2, 4, and 6 as having shorter wave lengths. If this beam of direct primary radiation is used to take a roentgenogram of the lower molar

Fig. 2.



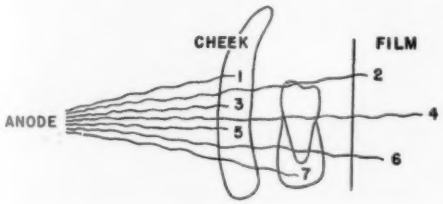


Fig. 3.

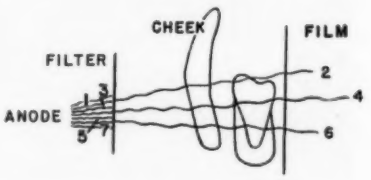


Fig. 4.

area, the action shown diagrammatically in Fig. 3 occurs. Rays 1, 3, 5, and 7, because of their long wave lengths, are absorbed by the skin of the patient's face or by the alveolar bone. Rays 2, 4, and 6, because of their shorter wave lengths, penetrate skin and bone and create a latent image on the dental x-ray film. Rays 1, 3, 5, and 7 are not useful radiations in exposing the roentgenogram.

When aluminum filters are placed between the radiation source and the tissue, the action shown diagrammatically in Fig. 4 occurs. Rays 1, 3, 5, and 7 are absorbed by the aluminum filters; rays 2, 4, and 6 reach the film. The total radiation reaching the film is approximately the same, whether filters are used or not, but the radiation absorbed by the patient's tissues is considerably reduced when filters are employed.

A number of dentists have asked the question: "Should the aluminum filters in an x-ray machine be changed every three months? I understand they become radioactive with use." The answer is "No." Aluminum filters do not lose their effectiveness any more than do sunglasses worn to protect the eyes from harmful rays of the sun.

PROJECT 2

Materials Used in Investigations.—A special lead backing device was constructed so that the radiation passing through the lead backing on the intraoral periapical film could be measured with a dosimeter. In essence, the backing device consisted of a lead barrier material of suitable thickness in which was prepared a rectangle suitable for insertion of an intraoral film. Through the use of this device, radiation measurements could be made as they passed through the dental film; areas beyond the limits of the dental film were made impermeable to the radiation used.

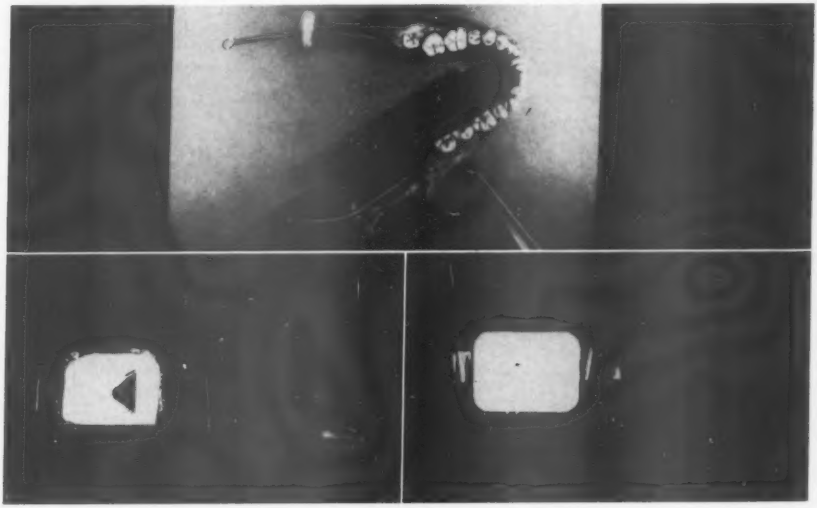


Fig. 5.

Methods of Investigation.—Wainwright and Villanyi[1] stated: "The skin at entrance, however, is not the only important part of the patient that is irradiated. The more penetrating beam from 90 KVP, of course, gives a higher skin dose at exit than does the less penetrating 65 KV beam. Also, the more penetrating 90 KV beam gives a higher rate of secondary radiation."

Measurements of radiation were made using 10 Ma., 2.5 mm. of added aluminum filtration, and voltages of from 45 KVP to 70 KVP. The special lead backing device previously described protected the dosimeter but allowed radiation that passed through the lead film backing to be measured. The x-ray cone tip touched the specimen on the buccal surface. Fig. 5 illustrates the shielding device.

Results of Investigation.—With 2.5 mm. of added aluminum filtration and a special lead backing device to protect the dosimeter from direct radiation, readings were recorded as the amount of radiation that passed through the lead film backing at the various kilovoltages. One-second exposures were used with the x-ray cone tip touching the specimen on the buccal surface. The readings were as follows:

45 KVP—10 Ma.—one-second exposure—0 mr
50 KVP—10 Ma.—one-second exposure—0 mr
55 KVP—10 Ma.—one-second exposure—1 mr
60 KVP—10 Ma.—one-second exposure—8 mr
65 KVP—10 Ma.—one-second exposure—17 mr
70 KVP—10 Ma.—one-second exposure—27 mr

The higher the voltage, the more radiation passed through the lead backing. Such radiation would have to be absorbed by the patient. In addition, greater amounts of secondary radiation are produced at the higher kilovoltages. As will be seen in the above figures, at 45 KVP no radiation was recorded through the lead backing; at 70 KVP, however, 27 mr passed through the lead backing. (Editor's note: Experimentally, a constant Ma. S. of 10 was selected. In practical usage, to obtain a constant film density, the milliampere seconds of exposure would be reduced as higher kilovoltages were employed. The values given by the author in the paragraph above cannot be directly related to usage in patients, since the radiation output as he progressed from 45 to 70 KVP is not constant.)

PROJECT 3

Materials Used in Investigations.—The films used were Eastman medium- and ultraspeed periapical films. For all recordings in Project 3, as well as in the foregoing Projects 1 and 2, a dried specimen with no overcoating to simulate soft tissue was used.

Methods of Investigation.—Medium- and ultraspeed films were exposed at one and two seconds of exposure, 10 Ma., and varying kilovoltages and filtration thicknesses. The radiation source-to-film distance was kept constant. Radiation measurements were recorded on the buccal surface as films were exposed. Films were processed at the same time by processing methods using standard time and temperature.

Results of Investigations.—The reader's attention is directed to Fig. 6. With 2 mm. of aluminum filter, the roentgenogram on the left (medium-speed) required a one-second exposure at 70 KVP and 10 Ma. to secure proper density. The roentgenogram on the right (ultraspeed) was exposed with 2 mm. of aluminum filtration at only 55 KVP and 10 Ma. in a one-second exposure to secure the same density. In the latter instance, the ultraspeed film reduced the necessary radiation by an amount equal to that produced by an added 15 KVP under other specified conditions.

Further evidence substantiating the advisability of using fast film is shown in Fig. 7. The roentgenogram on the left (medium-speed) required 750 mr of radiation to produce a proper density. The roentgenogram on the right (ultraspeed) required 210 mr to produce a similar density under constant exposure conditions. Thus, a savings of 540 mr of radiation was effected in a one-second exposure.

The use of the fastest possible film is the dentist's most effective way of reducing radiation to the patient and, incidentally, to himself.

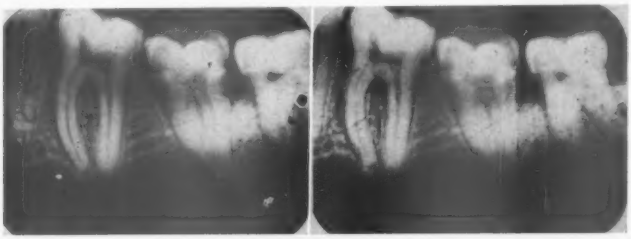


Fig. 6.

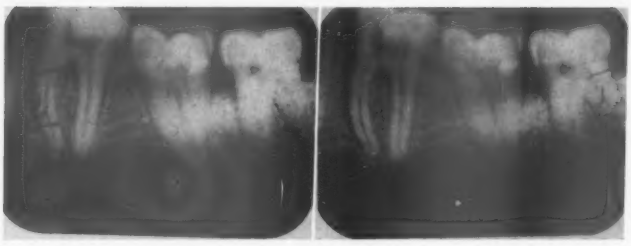


Fig. 7.

RECOMMENDATIONS

1. The output of each dental x-ray machine should be determined and means should be employed to keep the radiation that reaches the patient to a minimum.

2. All dental x-ray machines should be collimated appropriately.

3. All dental x-ray machines should be filtered with commercially pure aluminum to the maximum practical amount.

4. The fastest possible dental films should be utilized at all times.[3]

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ROENTGENOGRAPHIC LOCALIZATION OF UNERUPTED TEETH

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PERHAPS the most important reason for accurately determining the exact location or position of an unerupted tooth is the desire to avoid unnecessary tissue destruction through contemplated surgical or orthodontic procedures. Localization should mean more than simply locating a tooth. It is one thing to determine the mere presence of an unerupted tooth and quite another thing to determine its position and its relationships to other teeth and to important anatomic structures, such as the mandibular canal, the mental foramen, the maxillary sinus, the floor of the nose, and other landmarks. Localization implies establishment of a relationship between one tooth and another and between teeth and other antomic structures. Such knowledge is essential in the planning of surgical and orthodontic procedures involving unerupted teeth, as well as in the treatment of fractures, cysts, tumors, and other pathologic entities.

A number of roentgenographic procedures have been developed in an effort to provide this information. All make use of two or more films, and all utilize different angles of projection for each film. Such techniques are necessary since x-ray projections render the images of three-dimensional tissues on a two-dimensional film. The third dimension, that of depth, is missing from the single film. Multiple-film techniques are necessary for depicting the dimension of depth, even when stereoroentgenographic procedures are used. Stereoroentgenography can be helpful, but it has not been widely practiced by most dentists because the technique is detailed and time-consuming.

Multiple-film techniques are based on one or more principles of projection. Perhaps the most commonly used method is that of shifting the image of an object (the tooth) in a direction opposite that of tube movement through a change in horizontal angulation. The object that is farthest from the film will be moved more than an object that is closer to the film when viewed in two or more projections made from different angles (Fig. 1). However, all these techniques require a certain amount of imagination and mental gymnastics on the dentist's part, and often the dentist is not sure of his interpretation.

The technique that utilizes two or more projections at right angles to each other leaves the least doubt in the viewer's mind and demonstrates most accurately the dimension of depth and the relationships of structures.

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It is possible to project x-rays through a tooth at right angles to its long axis in a buccolingual, labiolingual, buccopalatal, or labiopalatal direction. This is the technique commonly used in periapical studies. Then, if a second film is exposed, with x-rays directed at approximately right angles to the rays used in the periapical projection, the true relationship of structures will be visualized. When the regions of the upper posterior teeth or of any of the lower teeth are

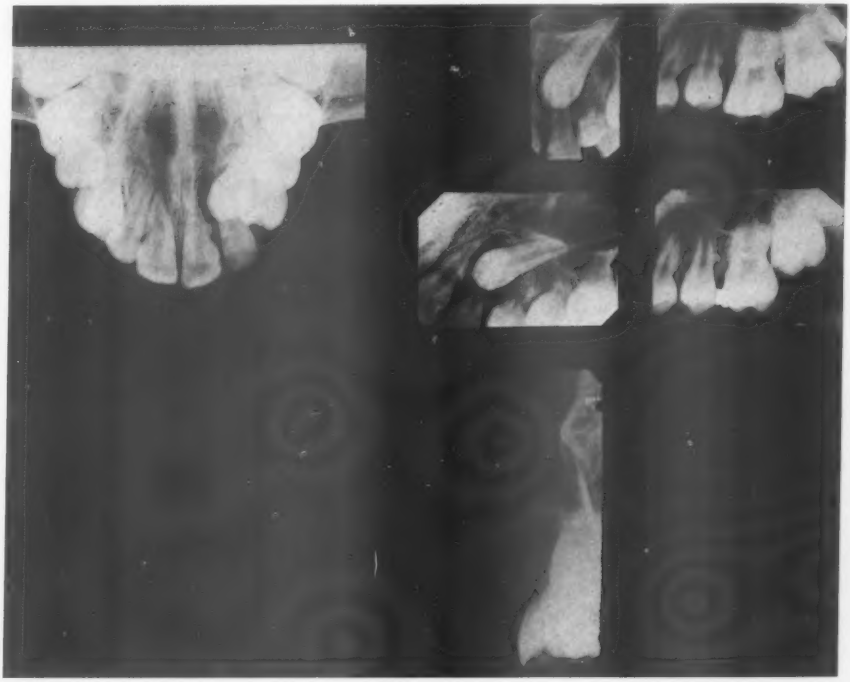


Fig. 1.—Several projections of the unerupted cuspid fail to reveal its location clearly until the anterior profile projection is made. The tooth is revealed to be in a position anterior and superior to the roots of the incisors.

to be studied, the second projection is usually made with the x-rays directed through the teeth from the apices to the incisal or occlusal surface. The images of the teeth in normal position are observed to have an "end-on" appearance (Figs. 2, 3, and 4).

Certain difficulties are encountered in making such projections of most upper anterior teeth because of the inclinations of these teeth. A line along the long axes of the teeth, extended apically, would come out at the vertex of the skull. Projection of x-rays along such an axis would, of course, pass through many skull and facial structures. This renders the images of the teeth very indistinct unless intensifying screens are used with an intraoral cassette and grid.

Unless such an angle of projection is used, the location of unerupted teeth can be very misleading. The usual "occlusal" film is exposed to x-rays projected at an angle of 45 to 65 degrees to avoid superimposing an image of the

Fig. 2.

Fig. 3.

Fig. 2.—Periapical and topographical projections show the unerupted premolar to be in a postion lingual to the roots of the first molar.

Fig. 3.—Periapical and occlusal projections reveal that the unerupted third molar is located more to the buccal side than is the second molar.

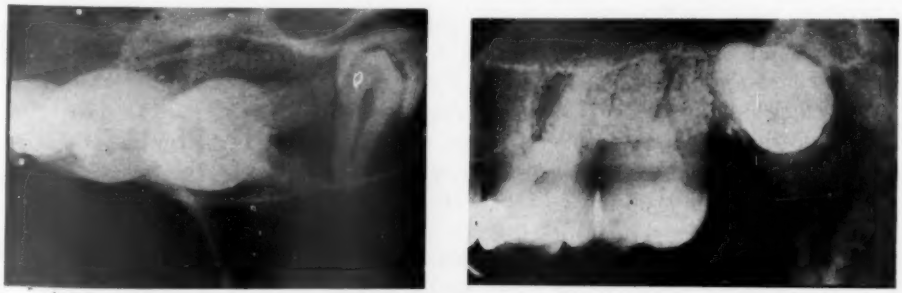


Fig. 4.—Periapical and occlusal projections reveal that the crown of the unerupted third molar is located toward the palate.

frontal bone on the area of interest rather than at a right angle to the film or parallel to and through the long axes of the teeth. This angle of projection does not permit an accurate analysis of relationships between the various structures. The projection made at an angle of less than 90 degrees to the film plane may be referred to as an occlusal or modified topographical view.

The principal purpose of this article is to describe a more readily useful procedure in which the x-rays are still directed at a right angle to the vertical plane of projection used to expose the periapical film. This procedure has proved to be particularly useful in localizing unerupted teeth in the anterior region of the maxilla. It may be referred to as the anterior profile projection. The area of the dental arch in which the unerupted tooth is located is determined clinically or by periapical projections. A second exposure is made with a standard dental film or an occlusal type of film. The film is placed extraorally on the side of the face closest to the region to be studied, with the lower

Fig. 5.—Position of film for anterior profile projections. Film of the conventional periapical type or of the occlusal type may be used.

film border 3 to 4 mm. below and parallel to the incisal plane. When the anterior region of the mandible is to be studied, the upper border of the film should be positioned 3 to 4 mm. above and parallel to the incisal plane of the mandibular incisor teeth. The anterior film edge should extend not more than 3 or 4 mm. in front of the labial surface of the anterior teeth. When the central and lateral incisor areas are to be studied, the film should be parallel to the midsagittal plane of the head (Fig. 5). It may be retained in position by a film block on which the patient closes his teeth. It also may be held by the patient's hand, but this increases the possibility of movement. Other mechanical devices also may be used to position and retain the film. The x-rays are projected at a right angle to the midsagittal plane and the film and parallel to the incisal or occlusal plane (Fig. 6). The central ray is passed through the apices of the incisor teeth. If the cuspid region is to be studied, the x-rays are projected tangentially to the curve of the dental arch in the cuspid region and the film is placed so that the x-rays can strike it at right angles (Fig. 7).

Except for this change, the technique is the same for the anterior and cuspid regions. Fig. 8 illustrates the location of an abnormality in the mandibular lateral incisor area.

With this technique, the anterior portion of the maxilla, including the nasal spine and the floor of the nose, the incisal edges of the anterior teeth, and some of the posterior teeth may be visualized. The labial or palatal position

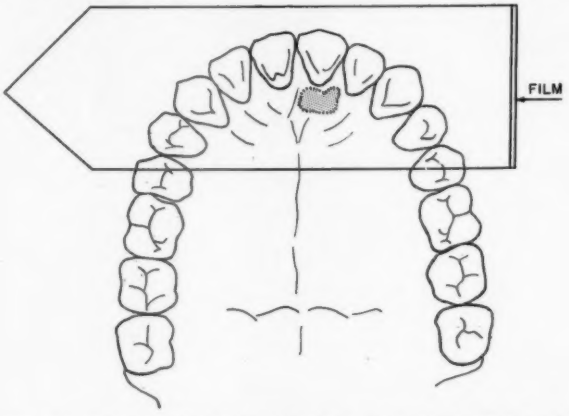


Fig. 6.—Diagram of film–ray–tooth relationship when the central and lateral incisor areas are examined by the technique described.

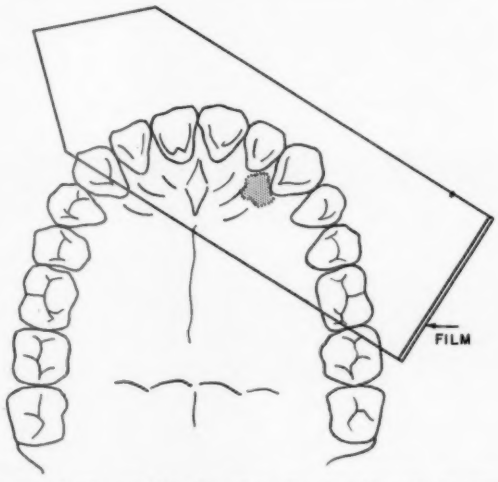


Fig. 7.—Diagram of film–ray–tooth relationship when the cuspid area is examined by the technique described.

of unerupted teeth may be determined readily and without doubt. Unerupted cuspids and supernumerary teeth are frequently found in this region of the maxilla (Figs. 9 and 10). This technique is especially useful for visualizing unerupted teeth which are labial or apical to the roots of normally

Fig. 8.

Fig. 9.

Fig. 8.—The periapical and topographical projections do not clearly reveal the location of the unerupted incisor and the odontoma. The anterior profile projection clearly indicates the labial position of the supernumerary tooth and the odontoma.

Fig. 9.—Periapical occlusal and anterior profile projections clearly show the unerupted cuspid to be located palatal to the roots of the incisors.

erupted teeth. Unerupted teeth that are positioned lingually are not as readily visualized because of the density created by the other superimposed dental structures.

Dental film of standard size can be used very satisfactorily for these projections. The tissues to be penetrated by the x-rays are, of course, thicker and more dense than in the usual periapical projection of the upper or lower

Fig. 10.—The anterior profile projections are necessary to demonstrate clearly the position of the unerupted cuspid.

incisor areas. Therefore, in order to penetrate these tissues more effectively, it is desirable to increase the kilovoltage applied to the x-ray tube. If the kilovoltage cannot be varied on the dental x-ray unit, however, an alternative is that of increasing the exposure time in an effort to increase the density of the film. An exposure time approximately three to four times that required for the periapical projection will usually provide a satisfactory film for interpretation.

SUMMARY AND CONCLUSIONS

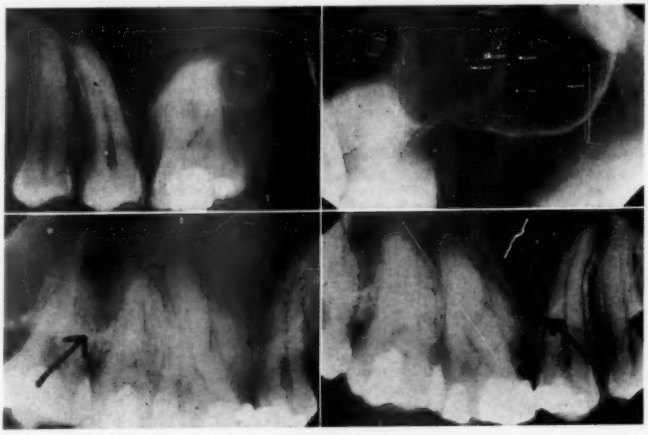
Any projection to aid in the localization of unerupted teeth should be considered a supplement to, rather than a substitute for, the recognized periapical projections. The anterior or lateral profile projection described is used to its fullest potential when a focal spot–film distance greater than 8 inches is employed. Standard periapical films are satisfactory, and no special equipment is needed. The mental gymnastics and misleading possibilities of some other techniques are avoided.

ROENTGENO-ODDITIES

Maxillary Sinus Abnormalities

Knowledge of the size and relationship of the maxillary sinus to the teeth is of utmost importance when extraction of the teeth or other surgical measures

A.



B.

Fig. 1.—*A*, A large maxillary sinus extending to the crest of the alveolar ridge and the tuberosity.

B, The maxillary sinus extending downward and occupying the space between the roots of the molar teeth and between the first molar and second premolar. The second premolar and the first and second molars appear to be pathologically involved.

Fig. 2.—Extraction of the first molar resulted in a portion of the lateral bony wall of the sinus adhering to the tooth. The opening into the sinus is not visible in the postoperative roentgenogram, since it is in the lateral wall and not the floor of the sinus.

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

Fig. 3.

Fig. 4.

Fig. 3.—An opening into the bony floor of the maxillary sinus which shows an abrupt interruption of the floor is usually indicative of a surgical defect of recent orgin.

Fig. 4.—A funnel-shaped opening into the bony floor of the maxillary sinus indicates the surgical defect to be of long standing.

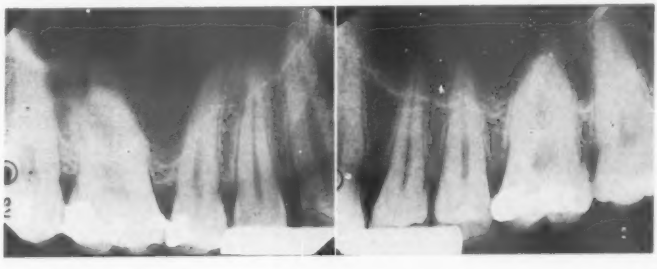


Fig. 5.

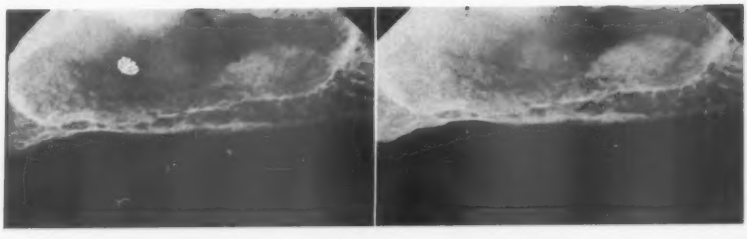


Fig. 6.

Figs. 5 and 6.—Mucoid retention cysts of the maxillary sinus originate in the mucosa of the sinus. They are characterized by a hemispherical or dome-shaped form with the base on the sinus wall or floor.

are contemplated. The accompanying illustrations are submitted by Donald T. Waggener, D.D.S., College of Dentistry, University of Nebraska, Lincoln, Nebraska.

ORAL PATHOLOGY

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A SOLITARY XANTHOMA OF THE ATTACHED GINGIVA

Report of a Case

A. P. Chaudhry, B.D.S., M.S., Ph.D., and R. J. Gorlin, D.D.S., M.S.,
Minneapolis, Minn.

INTRODUCTION

SEVERAL cases of xanthoma multiplex with involvement of the oral mucosa have been published in the literature,[1] but not a single case of solitary xanthoma originating in the attached gingiva has been reported so far. The present case is, therefore, of interest because of its rarity and because of the paucity of information concerning this lesion in the textbooks of oral pathology.

CASE REPORT

A 34-year-old white man noticed a firm, yellowish mass on his left lower gingiva between the lateral incisor and the cuspid tooth. The mass was entirely asymptomatic, and the patient was uncertain of its duration. He consulted his dentist, who referred him to a local physician. The physician excised the lesion and sent it to the pathology laboratory of the University of Minnesota School of Dentistry for histologic examination. The mass was descirbed as firm, yellowish, well demarcated, and adherent to the underlying periosteum.

Grossly, the specimen was circular, 8 mm. in diameter, and rather firm in consistency. The cut surface was yellow, smooth, and greasy. Microscopically, the tissue was covered with stratified squamous epithelium which was partly atrophic due to pressure from the underlying tumor mass. The rete pegs were flattened, with loss of papillae. Immediately beneath the basal-cell layer of the epithelium were present xanthoma or foam cells, giant cells, and fibroblasts with an admixture of inflammatory cells, particularly lymphocytes and histiocytes (Fig. 1). In multinucleated xanthoma cells, the nuclei were irregularly distributed, as in foreign body giant cells, or they were arranged near the center of the cell around a small, nonfoamy cytoplasm and surrounded by foamy cytoplasm (Touton giant cell) (Fig. 2). The picture was compatible with the diagnosis of xanthoma.

A thorough physical examination was essentially within normal limits. No other such lesions were found in the oral cavity or on the skin. Urinalysis was normal, without

From the Division of Oral Pathology, School of Dentistry, University of Minnesota.

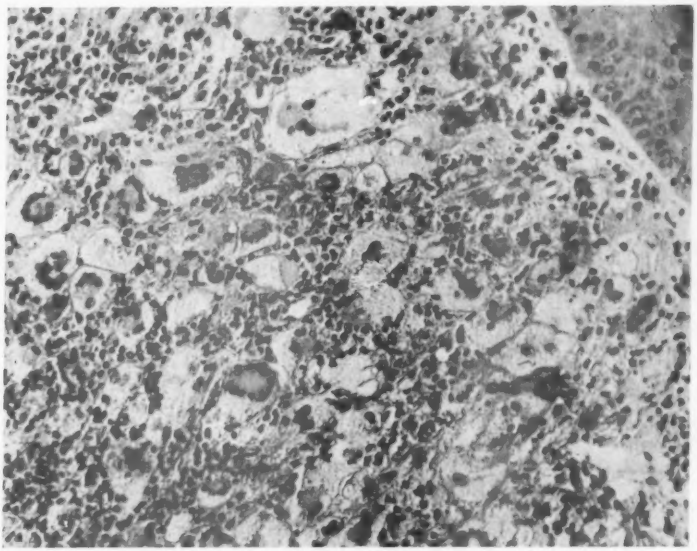

Fig. 1.—Photomicrograph showing close proximity of the tumor mass to the overlying flattened epithelium. Note foam cells, foreign body giant cells, and Touton giant cells.

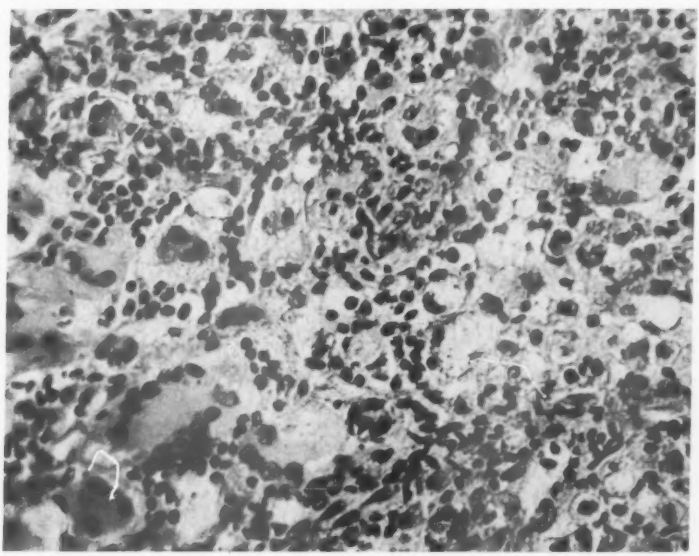

Fig. 2.—Photomicrograph (higher magnification) showing foam cells, foreign body giant cells, and Touton giant cells interspersed with inflammatory cells.

any evidence of diabetes mellitus. Hemglobin was 96 per cent; the white blood count was 10,600 c. mm.; blood cholesterol was 184 mg. per cent; and the basal metabolic rate was 21 per cent. Chest films and electrocardiogram were normal, and serology was negative.

The patient was last seen after one year, on Aug. 18, 1958, at which time there was no evidence of recurrence or of any new lesion in the mouth or on the skin.

DISCUSSION

The term "xanthoma" may be applied to a tumor of yellowish or yellowish pink color consisting of lipid-laden histiocytes (foam cells). Xanthoma may be solitary or multiple. Those who are interested in a detailed discussion of classification, clinical, histopathologic, and chemical studies of xanthoma should refer to other publications on the subject[2] or to the standard texts on dermatology.[3] No generally accepted classification of xanthomas exists. For the purpose of this report, it may suffice to say that multiple xanthomatosis, whether primary or secondary in nature, is most commonly associated with disturbances of cholesterol metabolism. On the other hand, solitary xanthomas, according to Bell,[4] are not associated with disturbance of cholesterol metabolism and appear to be neoplastic in nature. Karsner[5] considers them fibromas in which some of the cells become infiltrated with lipid. When lipid-filled cells are predominant, he prefers to call the tumors xanthoblastomas.

Since the tumor in the present case was solitary and was not accompanied by a disturbance of cholesterol metabolism, it would seem reasonable to consider it a benign neoplasm. The site of the lesion was unusual, since most solitary xanthomas occur in connection with tendon sheaths. It is highly probable that the tumor in this case might have originated from the periosteum.

SUMMARY

A case of solitary xanthoma arising from the attached gingiva in a 34-year-old white man has been described. No such lesion in the oral cavity has been previously reported.

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PAPILLARY CYSTADENOMA OF MINOR SALIVARY GLAND ORIGIN

Report of a Case

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INTRODUCTION

PAPILLARY cystadenoma of the minor salivary glands is a very uncommon neoplasm. Skorpil,[1] on reviewing the literature in 1941, found only eight cases of papillary cystadenoma of the salivary glands, including two of minor salivary gland origin. Of these, one occurred in the tonsillar region and the other in the soft palate. Subsequently, cases of papillary cystadenoma arising from minor salivary glands have been reported by Stout,[2] Hoboek,[3] Castigliano and Gold,[4] Vellios and Davidson,[5] and Brooks and associates[6] for a total of eight cases, to which we now add another. The available clinical data from nine cases have been summarized in Table I.

TABLE I

AUTHOR	YEAR	AGE (YEARS)	SEX	REGION	TREATMENT	RESULTS
1. Skorpil[1]	1941	78	M	Tonsillar	Excision	Two recurrences
2. Skorpil[1]	1941	28	M	Palate	Excision	—
3. Stout[2]	1946	50	F	Undersurface of tongue	Excision	—
4. Hoboek[3]	1949	45	F	Palate	Radiation	No recurrence, 2 years
5. Castigliano and Gold[4]	1954	52	F	Palate	Excision	—
6. Vellios and Davidson[5]	1955	—	—	Palate		
7. Brooks et al.[6]	1956	31	M	Palate	Excision	One recurrence
8. Brooks et al.[6]	1956	63	M	Palate	Excision	—
9. Chaudhry et al.	1958	64	M	Buccal parieties	Excision	No recurrence, 1 year

CASE REPORT

A 64-year-old white man was first seen in the office of a dentist on March 24, 1954. He was completely edentulous and had been wearing full dentures for several years. The dentures were loose and ill-fitting, and chronic irritation from them had caused denture-injury hyperplasia in both upper and lower sulci. Tags of hyperplastic tissue on the right and left buccal parieties were noted. The flanges of the dentures had produced areas of ulceration

From the Division of Oral Pathology, School of Dentistry, University of Minnesota, and the Division of Oral Diagnosis, School of Dentistry, Indiana University.

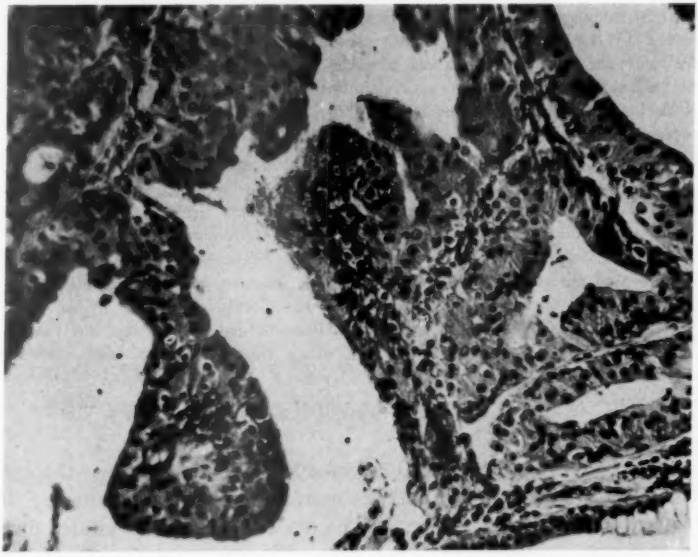

Fig. 1.—Photomicrograph showing ducts and dilated cystic spaces lined by pseudostratified columnar epithelium. Note the presence of goblet cells.

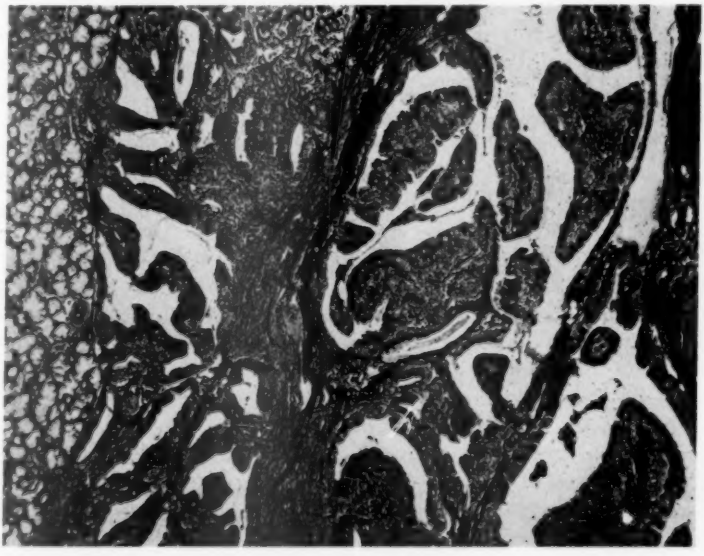

Fig. 2.—Photomicrograph showing papillary projections filling the spaces. Note close association of minor salivary glands with the neoplasm.

simulating squamous-cell carcinoma on both sides. Biopsies of the ulcerated areas, however, revealed only a nonspecific inflammatory process with no evidence of neoplasm. The patient was admitted to the hospital and, under general anesthesia, the hyperplastic tissue was completely removed. In the left buccal parieties, however, a well-circumscribed tumor mass, which was softer in consistency, was encountered. The mass was in close proximity to Stensen's duct but not associated with it. This piece was sent to the pathology laboratory for microscopic examination.

The specimen was covered with hyperplastic stratified squamous epithelium which showed acanthosis and spongiosis with broadening of the rete pegs. The tumor mass was separated from the epithelium by a scant connective tissue infiltrated with chronic inflammatory cells. The tumor was composed chiefly of dilated cystic spaces lined with pseudostratified columnar epithelial cells with a pinkish cytoplasm and oval, dark-staining, peripherally located nuclei. Interspersed between the columnar cells were goblet cells (Fig. 1). The spaces were filled with papillary projections which were also made up of tall columnar cells and were supported by a thin connective-tissue stroma. Close to the tumor, clusters of normal mucous glands were present, suggesting that the tumor had originated from minor salivary glands (Fig. 2).

DISCUSSION

As is evident from the small number of published cases, this neoplasm is extremely uncommon. No valid conclusions concerning its behavior, in terms of its benign or malignant nature, can be drawn from the available data. The data do indicate, however, that the tumor occurs more frequently in males (3:1) and that it has a predilection for the palate although other locations in the oral cavity are not immune to its occurrence.

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ENDODONTICS

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Captain Warren J. Hedman, Editor

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HISTOLOGIC EFFECTS OF VARIOUS DRUGS ON AMPUTATED PULPS OF RAT MOLARS

Marvin Kozlov, D.D.S., M.S., and Maury Massler, D.D.S., M.S.,** Chicago, Ill.*

INTRODUCTION

A NUMBER of histologic investigations of human and animal teeth have demonstrated that the injured pulp has a good potentiality for healing by the formation of a bridge of reparative dentine over the exposed portion of the pulp.[1-10] Reparative dentine formation has been seen repeatedly after vital pulp amputation, especially after the application of calcium hydroxide.[1-3] For a long time reparative dentine formation was thought to result from the action of the calcium ions. The studies of Miyamoto[7] and of Berman[8-10] suggest that a calcium salt is *not* essential for the formation of reparative dentine. Miyamoto and Berman found that dentine bridging occurred under noncalcium materials, such as wax, zinc oxide-eugenol, acrylic resin, and amalgam, placed on the amputated pulps of rat molars. The reparative dentine was quite similar to that found under calcium hydroxide, although quantitatively it was less. From this finding, it was assumed that the formation of reparative dentine under the site of amputation was a function of the pulpal cells themselves rather than of the medicament placed over them.

The number and types of agents tested, however, have been relatively narrow. There have been only a few attempts to test the effects of substances with widely different chemical, physicochemical, or pharmacologic properties.[11] In this investigation we sought to study the histologic response of the pulp to a number of specific drugs which have not previously been used on amputated

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These studies were aided by a contract, NR 105-046, between the Office of Naval Research, Department of the Navy, and the University of Illinois.

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**Professor and Head of the Department of Pedodontics.

pulp tissue, with the hope of clarifying the general pattern of pulpal healing as well as elucidating its specific response to specific drugs. The main objective was to see whether the pulp's tendency to bridge the gap could be accelerated.

REVIEW OF THE LITERATURE

Human Pulp Studies.—Histologic studies of pulpal healing and the preservation of the vitality of exposed human pulps by rational biologic methods were pioneered by Hess[12] and the Zürich School of Dentistry. From 1936 to 1949 they covered the exposed pulp with various calcium compounds. Their objective was to provide the injured pulp with a biologic wound dressing to allow the pulp to maintain its function and vitality. They had 85 to 90 per cent success. Hess[12] and Berman[9] have written extensive reviews of the literature of this era.

Glass and Zander,[1] in the United States, compared the capping of human pulps using calcium hydroxide and zinc oxide and eugenol. Calcium hydroxide showed rapid healing and bridging within four weeks. They found no reparative dentine under zinc oxide and eugenol. Instead, these pulps showed a chronically inflamed condition.

Englander, Massler, and Carter[13] described and clinically tested a "partial pulpotomy" technique. Carious, exposed pulps of young adults were partially amputated. A clot was allowed to form over each pulp, and either a calcium salt, an antibiotic, or one of a number of miscellaneous substances was applied over the blood clot. The cavity was then sealed with zinc oxide and eugenol and filled with zinc phosphate cement. These authors considered the presence of pain as the principal criterion of success or failure. Pain was present in only 11 per cent of the teeth. Calcium hydroxide mixed with methyl cellulose was the most effective of the calcium salts used. The use of antibiotics and silver nitrate resulted in a higher percentage of failures. This investigation demonstrated that clinical success in treating pulps is predicated less upon the type of agent used than upon the technique and care taken by the operator.

In 1957, James, Englander, and Massler[5] studied the histologic response of amputated human pulps to various calcium compounds and antibiotics used in the previous study. There was 43 per cent bridging under calcium compounds and 23 per cent bridging under antibiotics. Calcium hydroxide with methyl cellulose produced the highest percentage of dentine bridges. Bridging decreased as inflammation increased.

In general, histologic studies of human teeth suffer from a paucity of material, a great variability in pulpal responses, and the difficulty of running large series of experiments under controlled conditions. The teeth of rodents, therefore, have been used more recently for such experiments.

Rodent Pulp Studies.—O'Malley[6] studied the histologic reactions of amputated pulps in rat incisors and the effects of various substances on the reparative process. He found that the pattern of pulpal healing and zoning was basically the same, with the exception of zinc oxide and eugenol, no matter what material was placed on the injured pulp. He found that a zone of

dystrophic calcification (calcified fibrous tissue) always formed between the injured area and the normal pulp tissue within seven to fourteen days after surgery.

Because the rat molar is morphologically similar to the human molar, Berman[10] and Miyamoto[7] chose this tooth as their test object. Miyamoto[7] placed various calcium salts (calcium hydroxide, calcium carbonate, and tricalcium phosphate), silver amalgam, acrylic resin, silicate cement, zinc phosphate cement, and wax on the amputated pulp and observed their effects over periods of seven to twenty-eight days. Like O'Malley, he found little difference in the pulpal reaction under different medicaments. Reparative dentine bridges were formed under all noninjurious substances. Miyamoto concluded that the pattern of pulpal healing was a stereotyped one under noninjurious substances. Under injurious substances, such as acid cements, the pulp degenerated and became necrotic. Both Miyamoto and Berman found that the most important single factor in the production of a necrotic pulp was loss of the filling or marginal leakage.

Berman and Massler[10] analyzed the mechanism of pulpal repair in rat molars in greater detail. They compared the pattern of healing under calcium hydroxide and zinc oxide and eugenol. This study verified Miyamoto's conclusion that the response of the pulp was stereotyped and followed a definite sequence. The initial reaction (at seven days after amputation) was the formation of a zone of inflammatory cells immediately under the site of amputation. At fourteen days, a fibrous capsule appeared to surround the inflammatory area. The fibers were coarse and tended to calcify early. This primary calcific bridge or zone of dystrophic calcification was the most constant and characteristic feature of pulpal healing at fourteen days after amputation. Berman felt that it acted as an "organizer" for the pulpal cells to form dentine since new dentine formation began only after the primary dentine bridge was completed. The changes at fourteen days were therefore important indications of the progress of healing. At twenty-one and twenty-eight days after amputation the pulp showed four distinct zones: (1) the layer of medicament and blood on the surface, (2) a zone of superficial pulpal injury and degeneration, (3) the fibrous calcified bridge (Berman called this material the primary calcific bridge, Miyamoto called it the necrobiotic zone, and O'Malley referred to it as a zone of dystrophic calcification), and (4) the permanent dentine bridge.

Berman and Massler[10] found that the pattern of pulpal healing in the rat molar was the same, whether calcium hydroxide or zinc oxide and eugenol was used, with only minor differences. The differences were mostly a function of time. Healing was accelerated under calcium hydroxide, and under zinc oxide and eugenol it was slower. These differences were most obvious at seven and fourteen days after amputation. By twenty-one and twenty-eight days, the pattern of healing was very similar under both substances and only slightly different in rate.

Quigley[14, 15] performed similar pulp amputations in the molars of hamsters. In 1956 he studied the effect of zinc oxide and eugenol and calcium hydroxide on the pulp. He found that zinc oxide and eugenol caused an acute hyperemia,

abscess formation, and degeneration of odontoblasts. Calcium hydroxide initially gave good results, but after a period of time degeneration set in—even after a bridge of tubular dentine was laid down.

The difference between Quigley's results and those of Miyamoto and Berman might be due to filling leakage. The fact that there was bridging under calcium hydroxide but that there was also chronic inflammation leads one to believe that filling leakage might have retarded the complete resolution of the initial inflammatory process.

In his second study Quigley[15] sought to determine the effect of a blood clot over the exposures of hamster pulps. He covered the amputated pulps with gelatin sponge alone or gelatin sponge saturated with 1:1,000 epinephrine. At two hours there was no difference between pulps treated with the gelatin sponge with or without epinephrine. The specimens at seventy-five days all showed severe degenerative changes.

Kiryati[11] tested the effects of hydrocortisone alone and hydrocortisone mixed with antibiotics on infected pulps of rat molars. Controls filled with zinc phosphate cement at fourteen days showed the formation of a "coarse fibrillar osteoid-like structure" demarcating the necrotic area from the rest of the pulp. This was undoubtedly the same material described by O'Malley, Miyamoto, and Berman. Pulps treated with hydrocortisone showed the same picture as that seen under zinc phosphate cement. The combination of hydrocortisone with calcium salts did not produce as favorable a reaction. Many pulps became necrotic. When neomycin and bacitracin were added, the pulps survived more normally. In general, combinations of hydrocortisone and antibiotics favored the survival of the pulp after injury and infection. Combinations with zinc oxide and eugenol also produced favorable pulp responses.

The preceding studies suggest that the amputated pulps of rodents tend to heal in a stereotyped manner if the agent applied is noninjurious and the fillings do not leak and are not lost. The purposes of our study were to test this hypothesis, using a variety of drugs, and to see which agents could significantly speed up or retard the rate of pulpal healing.

METHODS AND MATERIALS

This study utilized 132 molar teeth in sixty-six hooded rats, 60 to 90 days of age.

Each animal was anesthetized by intraperitoneal injections of 1 per cent sodium pentobarbital (Nembutal), 0.5 c.c. per 100 grams of body weight. The animal was then placed on its back on an operating table similar to that devised by Maurice and Schour.[16] The mouth was kept open as described by Berman.[8] The teeth were dried with an air jet and cotton pellets. During the operation, both air and cotton were used to keep the area dry and clean. At times, trimmed paper points were placed in the buccal sulcus to maintain dryness. These measures were not completely effective, however, and saliva occasionally leaked into the operation site.

A binocular dissecting microscope with a magnification of 9× was used for the operation. An ordinary dental handpiece and contraangle, a No. 33½ inverted cone, and half-round burrs were used to cut the cavities and amputate the pulps. Dental explorers and other dental instruments were modified at their ends and sharpened for specific procedures.

The first maxillary molar of the rat was chosen, since it was the largest of the molar teeth and had a larger pulp chamber. Access to this tooth was easier than to the other molar teeth.

Precautions were taken to maintain an adequate airway during the operation and to cleanse the nasal passages frequently with mild suction. The mortality rate was approximately 8 per cent.

Cavity Preparation and Pulp Treatment.—The occlusal surface was opened approximately in its center and a cavity was prepared slightly larger than the diameter of a No. 33½ inverted cone burr. A large exposure resulted when the burr was allowed to penetrate the pulp. Small exposures were made by stopping the burr just before exposure and removing the remaining dentine with hand instruments. Bleeding was controlled by sponging only.

As Berman[8] demonstrated, blood or debris can block the action of a medicament if they come between the agent and the pulp. Therefore, special attempts were made to remove blood and dentine debris before applying the drug. Drugs in liquid form were applied to the pulp by saturating them on Whatman filter paper and sealing them with amalgam. The solid or semisolid medicaments were applied to the pulp in the form of pastes. The medicaments were sealed with silver amalgam.

Occasionally, a blood mass or spicule of dentine was later found between the drug and the pulp. This interfered with the drug-pulp interaction. In such cases, the analysis was based on areas of the pulp in direct contact with the drug.

Powdered Purina Chow and water were fed ad libitum. Powdered food was used to reduce the number of fractured fillings.

The animals were sacrificed at one hour or at fourteen days after the operation by anesthetization with ether and decapitation. The heads were skinned and quickly dissected, after which the maxillae were split down the midline to ensure adequate penetration of fixative and decalcifying agent.

The maxillae were fixed in Zenker's formol solution, washed, dehydrated, embedded in paraffin and sectioned serially at 10 microns. The sections were stained with hematoxylin and eosin stain and Mallory's connective tissue stain.

Drugs Used.—The following substances were applied to the amputated pulps:

A. *Anti-inflammatory agents**

1. Chlor–Trimeton maleate (Schering), 100 mg. per cubic centimeter

*For the exact composition of each of the substances used, see original Master's Thesis by Kozlov, University of Illinois, 1959.

2. Cortisone acetate (Schering), 25 mg. per cubic centimeter
3. Hydrocortisone acetate U.S.P. (Upjohn), 25 mg. per cubic centimeter

B. *Vasoconstricting agents*
1. Kutapressin (Kremers–Urban)

C. *Dyes*
1. Malachite green (0.1 per cent solution in saline)
2. Gentian violet (0.1 per cent solution in saline)
3. Carmine red (0.1 per cent solution in saline)

D. *Irritants*
1. Concentrated formaldehyde (37 per cent solution in water)
2. Sodium hydroxide (50 per cent solution in water)
3. Acetic acid (50 per cent solution in water)
4. Xylene (Mallinckrodt)
5. Metacresylacetate (Cresatin, Merck, Sharp & Dohme)
6. Calcium chloride (crystalline)

E. *Oxygenating agent*
1. Hydrogen peroxide 30 per cent (Superoxol, Merck)

F. *Mixtures of Agents*
1. Calcium hydroxide and Cresatin

G. *Controls*
1. Zinc oxide and eugenol
2. Calcium hydroxide
3. Silver amalgam (without paper)
4. Whatman filter paper alone, under the amalgam filling
5. Open cavities

Zinc oxide and eugenol, calcium hydroxide, and silver amalgam were placed on the pulp for purposes of comparison, since they had been used by others over amputated pulps of rat molars.[7, 8]

Because it was the vehicle for most of the drugs used in this study, Whatman filter paper was placed on the pulp under amalgam fillings in order to test the effect of the paper alone. In addition, eight pulps were amputated and left open to the oral cavity for one hour (seven specimens) or fourteen days (one specimen).

FINDINGS

In general, four types of pulpal reactions could be distinguished under the drugs used in this study (Table I and Fig. 1).

Group A. Good Reparative Response.—These were characterized (at fourteen days) by a small zone of inflammation, well encapsulated by a narrow but dense layer of calcified fibers under which a wide bridge of regular reparative dentine was formed centrally and laterally to bridge the site of amputation completely. The pulp below was normal with no inflammatory cells visible. A slight increase in collagen fibers could be seen in sections stained with Mallory's

TABLE I. PULPAL REACTIONS TO DIFFERENT DRUGS (AT FOURTEEN DAYS AFTER AMPUTATION)

DRUGS USED	NO. OF SPECIMENS	ZONE I INFLAMMATORY REACTION	ZONE II DEFENSE AND REPAIR	ZONE III PULP
Group A. Good Reparative Reaction				
Rapid Repair				
Cortisone	12	None	Wide reparative dentine bridge directly under site of exposure	Pulp below dentine bridge relatively normal
Moderately Advanced Repair				
Xylene	3	Inflammatory zone small; surface may be coagulated or degenerated	Inflammatory zone well encapsulated by calcified fibrous band and thick layer of central reparative and lateral secondary dentine	Slight increase in collagen fibers
Amalgam (silver)	3*			
Chlor-Trimeton	18			
$Ca(OH)_2$ with Cresatin	2			
Slightly Advanced Repair				
Hydrocortisone	7		Layer of reparative dentine thin; calcified fibrous material; fibrous peripheral dentine only	
$Ca(OH)_2$	2†			
Group A'. Good Defensive Reaction, Slow Repair				
ZOE	11†	Narrow inflammatory zone	Dense band of calcified fibers walling off inflammatory zone but no central formation of reparative dentine; small amount of secondary dentine on adjacent walls	Pulp is relatively normal
Group B. Fair Defensive Reaction, No Repair				
Encapsulation by Calcified Fibrous Material				
Superoxol	7	Large zone of inflammation	Inflammatory zone encapsulated by band of loose, calcified fibers; no reparative dentine formation centrally; very slight amount formed laterally	Pulp is relatively normal but contains engorged blood vessels
Cresatin	3			
Encapsulation by Fibrous Tissue Alone				
Acetic acid	3	Degeneration and necrosis on surface of inflammatory zone, prominent	Fibrous capsule loose and not calcified	Fibrous dentine formation on walls of root canals
$CaCl_2$	3			
Formol-cresol	‡			
Group C. Inflammatory Reaction, Poor Defense				
Moderate Inflammatory Cell Penetration—Poor Encapsulation				
Carmine red	2	Penetration of inflammatory cells to mouth of canals and beyond	Poor encapsulation; loose fibers only	Pulp fibrotic; calcified fibrous material formed along walls of canals
Gentian violet	2			
Filter paper	4			

(Continued on following page.)

TABLE I—CONT'D

DRUGS USED	NO. OF SPECIMENS	ZONE I INFLAMMATORY REACTION	ZONE II DEFENSE AND REPAIR	ZONE III PULP
Severe Inflammatory Cell Penetration—No Encapsulation				
Kutapressin	5	Deep penetration of inflammatory cells into canals; wide zone of coagulation and degeneration on surface	Little or no encapsulation	Severe fibrotic degeneration of remaining pulp
Malachite green	2			
Concentrated formaldehyde	6			
Formaldehyde	‡			
Cresol	‡			
Zinc phosphate cement	§			
Group D. Destructive Reaction, No Defense				
NaOH	3	Inflammatory cells penetrated throughout necrotic pulp	No encapsulation	Degenerated
Silicate cement	§			
Lost fillings	13			
Unoperated controls	21			
Total	132			

*Findings similar to larger series reported by Miyamoto.[7]
†Findings similar to larger series reported by Berman.[8]
‡From Mansukhani.[17]
§From Miyamoto.[7]

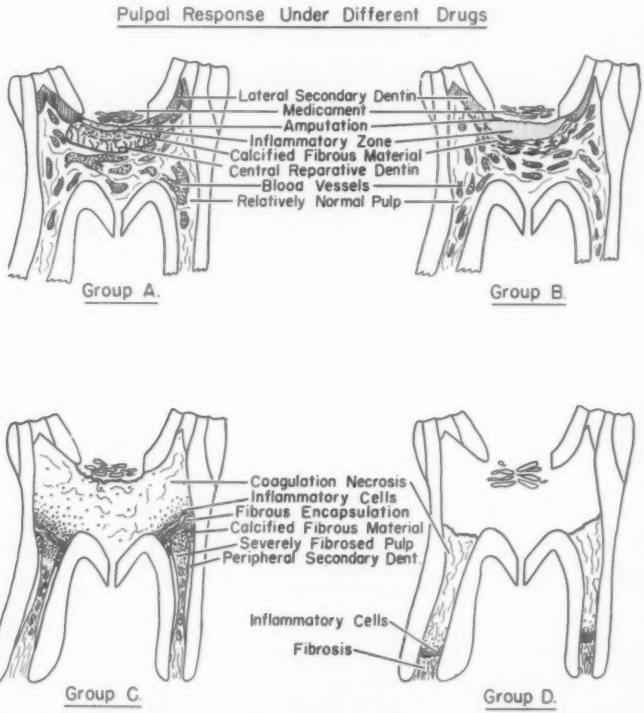

Fig. 1.—Diagram illustrating different types of pulpal reactions under different drugs.
Group A: Good reparative reaction (complete bridging).
Group B: Fair defensive reaction, no repair.
Group C: Inflammatory reaction, poor defense.
Group D: Destructive reaction, no defense.

connective tissue stain. This type of reaction was observed under cortisone, xylene, silver amalgam, Chlor–Trimeton, calcium hydroxide with metacresylacetate, hydrocortisone, and calcium hydroxide, the amount of reparative dentine decreasing in that order (Fig. 2).

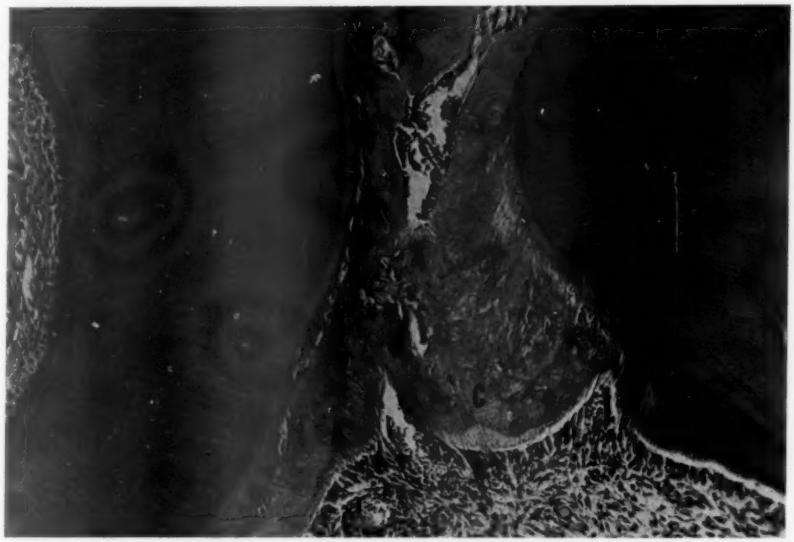

Fig. 2.—Photomicrograph showing good reparative reaction under Xylene (Group A) at fourteen days. Note the formation of lateral secondary dentine *(A)* on the chamber walls and its union beneath the exposure with the central reparative dentine *(C)* to form a complete bridge. The inflammatory zone is not prominent. The pulp below is normal. (Hematoxylin and eosin stain. Magnification, ×250; reduced 3/10.)

Group A′. Good Defensive Reaction, Moderate Repair.—Within Group A there was a subgroup in which reparative dentine formation was less evident than in the group above. The zone of inflammation was well contained within a dense capsule of calcified fibers, and a small amount of secondary dentine was formed along the walls lateral to the amputation site, but no reparative dentine was apparent centrally (at fourteen postoperative days). The pulp was relatively normal (Fig. 3).

This reaction was seen under zinc oxide and eugenol and in general was considered to be the "average" picture of pulpal repair at fourteen days (Fig. 3). The reaction observed in Group A above was considered to be "advanced." (See also Berman.[8]) This category did not really differ from the preceding, since both constituted a successful healing. Group A revealed a rapid healing process, while Group A′ shows a slower, "average," healing rate. According to Berman[8] differences between the reaction under calcium hydroxide (Group A) and zinc oxide and eugenol (Group A′) were apparent at seven and fourteen days but disappeared at twenty-one and twenty-eight days. Group B below

differed from these two groups in that healing (by complete bridging) was questionable and even the defensive reaction sometimes broke down.

Group B. Fair Defensive Reaction, No Repair.—This group was characterized by the presence of a large zone of inflammation with some signs of degeneration or necrosis at its outer surface. The area was encapsulated by a loosely woven, wide fibrous capsule which was calcified, but there was no evidence of reparative dentine formation centrally and very little laterally. The pulp below was usually relatively normal but contained many engorged blood vessels. This reaction was seen under hydrogen peroxide 30 per cent and under metacresylacetate (Table I).

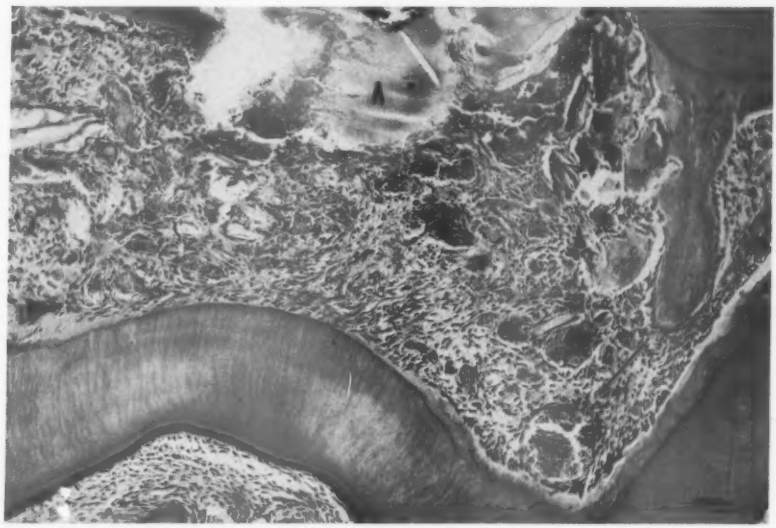

Fig. 3.—Photomicrograph showing the reparative reaction under zinc oxide and eugenol (Group A') at fourteen days. Note the normal pulp beneath the medicament (*A*). There is some increase in vascularity (arrow) and secondary dentine formation lateral to the site of amputation but no central dentine formation at fourteen days. (Hematoxylin and eosin stain. Magnification, ×160; reduced 9/10.)

Under some materials (acetic acid and calcium chloride) the fibrous capsule was loosely woven and not calcified, and the underlying pulp showed evidence of fibrosis (Fig. 4). This reaction was also seen under formol-cresol by Mansukhani.[17]

Group C. Inflammatory Reaction, Poor Defense.—Histologic sections obtained from this group showed a large zone of inflammatory cells penetrating deeply into the underlying pulp through a poorly formed and diffuse layer of collagenous fibers. There was no capsule formation. The tissue next to the medicament was coagulated or degenerated, and the pulp tissue deep in the canal was fibrotic and already degenerating.

This reaction was seen under the dyes, Kutapressin, formaldehyde, and (surprisingly) also under the paper controls. It should be remembered that the latter were covered with amalgam only, and the possibility of leakage cannot be excluded.[17] A similar reaction under zinc phophate cement was described by Miyamoto.[7]

Fig. 4.—Photomicrograph showing the loose fibrous capsule formation *(A)* typical of the medicaments in Group C at fourteen days. Note the absence of reparative dentine and the extension of the collagenous fibers into the pulp canals. (Mallory's stain. Magnification, ×160; reduced 3/10.)

Group D. Destructive Reaction, No Defense.—The pulp was completely necrotic under sodium hydroxide, under unfilled cavities, and when the fillings were cracked or lost (Fig. 5).

DISCUSSION

It was evident that the rat molar pulp tends to follow a stereotyped pattern of healing. There was first a strong inflammatory reaction, followed by a fibrous encapsulation of the inflammatory cells (defense reaction). The fibrous capsule soon became calcified. This was then followed by the formation of a new dentine bridge (healing).

This investigation was designed to study the histologic response of the amputated pulp of rat molars to a number of specific drugs for the purpose of better defining those conditions or pharmacologic actions which might promote or retard healing.

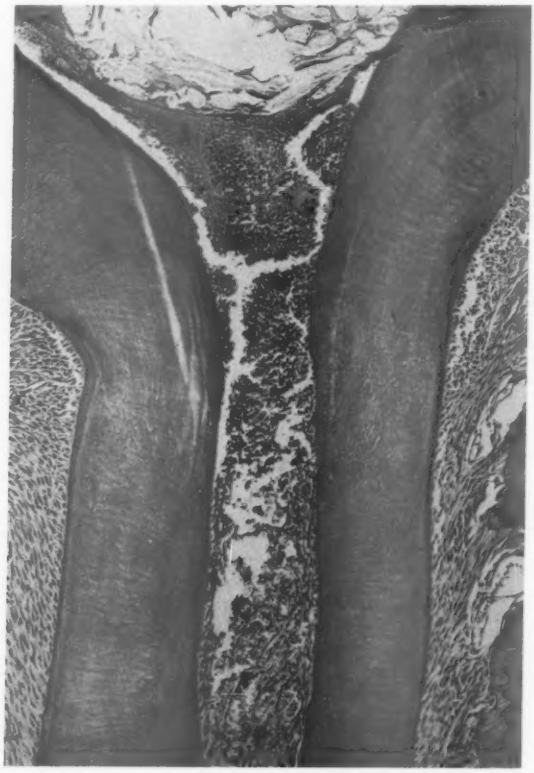

Fig. 5.—Photomicrograph showing deep infiltration of inflammatory cells and pulp destruction under sodium hydroxide (Group D) at fourteen days. Notice the pulpal coagulation at *A* and the small amount of pulp remaining in the apex (*P*). (Hematoxylin and eosin stain. Magnification, ×150; reduced 9/10.)

If the pulpal reaction under the filter paper controls is taken as "average," then the materials in Groups A and B might be considered as having accelerated the reparative reaction at fourteen days. However, since the controls showed less healing than substances which are known to be injurious to the pulp (acetic acid, formol-cresol), it is more likely that the filter paper (or some substance in the paper) causes an unfavorable reaction. For this reason, and on the basis of previous findings by Miyamoto[7] and Berman,[8] it would be better to compare the pulpal reactions with those under zinc oxide-eugenol and calcium hydroxide as "controls."

If this base line is accepted, then cortisone, xylene, silver amalgam, and Chlor–Trimeton each produced some acceleration in healing. The question now arises: "what properties of these substances could be responsible for this acceleration?" Unhappily, there are no properties common to all the substances in this group. Cortisone and Chlor–Trimeton are anti-inflammatory agents. Silver amalgam is a heavy metal combination not noted for any particular tissue reaction. It was quite surprising to find that amalgam capping produced pulpal healing equal to or better than calcium hydroxide. Xylene, which is an irritant and causes inflammation in connective tissue, produced practically no inflammation when placed on pulp tissue and led to a very successful bridging of the pulp! This study re-emphasizes the finding that calcium hydroxide is not necessary for the formation of secondary or reparative dentine in the rat molar pulp.

In general, specimens that showed less inflammation also showed better repair. This supports similar findings by James and associates[5] in human teeth. The hypothesis that irritation causes inflammation and that repair then follows was not supported by this series.

Hydrocortisone, which is supposed to be a more potent suppressor of inflammation and fibroplasia than cortisone,[18, 19] did not act this way on the pulp. Actually, hydrocortisone caused less rapid healing of the pulp than cortisone.

It was interesting to note that the mixture of calcium hydroxide and metacresylacetate was a greater stimulant to dentine formation than calcium hydroxide alone. Metacresylacetate (Cresatin) is a potent antiseptic and anodyne. Schilder and Amsterdam[20] found it to be nonirritating to connective tissue.

This investigation could not establish a relationship between pharmacologic action and pulp reaction. It is not yet clear which properties will stimulate defense and healing of the pulp. Stimulation of healing by bridging of the amputated area does not appear to be specifically related to the action of calcium ions, hydroxyl or hydrogen ions, anti-inflammatory or irritant substances, vasoconstrictors, or oxygen. It is possible that other concentrations of these agents than those used in this investigation might enhance or retard pulpal healing to different degrees. This hypothesis must be tested by additional experimentation.

A review of the excellent paper by Schilder and Amsterdam[20] made it apparent that the reaction of the pulp to a variety of drugs is not necessarily the same as the reaction of loose areolar connective tissue. Therefore, the living pulp must continue to be the test object in investigations of pulp-capping materials.

SUMMARY AND CONCLUSIONS

This investigation was designed to study the effects of various drugs on the pattern and rate of healing of amputated pulps in rat molars. The pulps of 111 maxillary first molars in sixty-six hooded rats 60 to 90 days of age were amputated with small burrs, and various medicaments were applied on saturated pieces of filter paper or as pastes and covered with silver amalgam fillings.

Twenty-one teeth were used as unoperated controls. The animals were sacrificed fourteen days later, and the pulps were studied in serial histologic sections.

This study confirmed the pattern of pulpal healing as described by Berman[10] and Miyamoto.[7] First there was a zone of inflammatory cells of varying widths immediately under the site of amputation and medicament. If a favorable reaction occurred, the inflammatory zone was walled off or encapsulated by a layer of calcified fibrous tissue. This was termed the defensive reaction, and its presence was essential before the wound could be healed by a bridge of dentine. If the calcified capsule was incompletely formed or absent, infiltration of polymorphs into the underlying pulp occurred, with fibrosis or necrosis following.

Healing occurred by the formation of secondary dentine by old odontoblasts along the walls lateral to the site of amputation and by the formation of reparative dentine centrally by new odontoblasts under the calcified fibrous layer. Under optimal conditions, these joined to form a complete dentine bridge over the normal pulp in fourteen days.

The reactions to the drugs used in this study could be classified into four groups:

Group A. Good reparative response: The pulpal reaction in this group was characterized by a small inflammatory zone well encapsulated by a dense calcified band and good secondary and reparative dentine formation. Bridging was well advanced or complete under cortisone, xylene, silver amalgam, Chlor–Trimeton, calcium hydroxide with metacresylacetate (Cresatin), hydrocortisone, and calcium hydroxide, the amount of reparative dentine decreasing in that order. Reparative dentine was just beginning to form under zinc oxide and eugenol.

Group B. Fair defensive reaction, no repair: Specimens in this group showed a moderately large inflammatory zone which was fairly well encapsulated by a fibrous layer. The latter was calcified under 30 per cent hydrogen peroxide and metacresylacetate but uncalcified under acetic acid and calcium chloride. There was no evidence of reparative dentine formation under the fibrous capsule, and very little secondary dentine formed laterally. However, the pulp was relatively normal.

Group C. Inflammatory reaction, poor defense: The defense mechanism in this series was depressed, and only occasional fibrous strands were visible under a large zone of degenerated and coagulated pulp tissue. Inflammatory cells penetrated deeply into the underlying pulp which was already undergoing fibrotic changes at fourteen days. These changes were less severe under dyes, filter paper, Kutapressin (a vasoconstrictor), and more severe under concentrated formaldehyde solution.

Group D. Destructive reaction, no defense: The pulp was as completely degenerated under sodium hydroxide as it was when the filling was lost or when the pulp was deliberately left exposed to the oral cavity.

The agents used in this study did not modify the basic pattern of pulpal healing, but they did affect the rate of healing by accelerating or depressing the defensive reaction. It is not yet clear which pharmacologic or chemical properties will stimulate a strong reparative reaction or a rapid degenerative process. Stimulation of bridging over the amputated area did not appear to be specifically related to the presence of calcium ions, hydroxyl or hydrogen ions, anti-inflammatory or irritant substances, vasoconstrictors, or oxygen.

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RESEARCH

THE EFFECTS OF DESALIVATION ON PERIODONTAL TISSUES OF THE SYRIAN HAMSTER

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AN INCREASING amount of research is being conducted on the physico-chemical properties of saliva in an attempt to define its role in the etiology of dental caries. Hakusima[1] suggested that saliva contains an active factor, possibly a hormone, which is related to the health of the periodontal tissues. However, no valid confirmation of the above assertion is yet available.

It has been repeatedly demonstrated that the removal of the salivary glands of experimental animals increases significantly the incidence of dental caries.[2-4] There has been no definitive study, however, on the effect of desalivation upon the periodontal tissues. Weisberger and associates,[5] while studying the relationship of desalivation to dental caries, frequently observed periodontal lesions in white rats.

The objectives of the present study were to determine the effects of the removal of the major salivary glands upon the periodontal tissues of the Syrian hamster and to observe the effects of diets of different composition and consistency upon the incidence of periodontal lesions.

EXPERIMENTAL

Sixty Syrian hamsters, 35 days old, were divided into six groups of twelve, ten, ten, nine, eleven, and eight animals, respectively. The animals were distributed into groups that were comparable with respect to sex and age.

The major salivary glands of the animals of Groups 2, 4, and 6 were removed by the method of Cheyne.[6] The animals of Groups 1, 3, and 5 served as the intact controls for the desalivated animals of Groups 2, 4, and 6, respectively. The subjects of Groups 1 and 2 were maintained on a high-carbohydrate

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finely powdered ration 4,[7] while those of Groups 3 and 4 were given laboratory chow pellets. The hamsters of Groups 5 and 6 received a finely ground laboratory chow. The experimental design is given in the first four columns of Table I.

All animals were kept in individual wire-bottom cages in a temperature-controlled animal house and were given their respective diets and water ad libitum. At the end of the twelve-week experimental period, all the animals were decapitated under ether anesthesia. The heads were removed and were kept in 95 per cent alcohol for forty-eight hours, after which they were skinned. The number and extent of the periodontal lesions, in both soft and calcified tissues, were evaluated by the method of Gupta and Shaw.[8] After the gross evaluation of the lesions, jaws from representative specimens were decalcified, sectioned, and stained with hematoxylin and eosin.

RESULTS

Gross Observations—Weight Change.—The desalivated animals showed a decline in body weight immediately after removal of their salivary glands. These animals started gaining weight four to five days postoperatively. The final weight of the desalivated animals was 10 to 15 grams lower than that of their controls. The desalivated animals became irritable following removal of the salivary glands and remained so for four to five weeks.

There were no significant differences in the body weights of the animals maintained on laboratory chow pellets and on the ground laboratory chow. On the other hand, the animals maintained on either the chow pellets or the ground chow were 5 to 10 grams heavier than those on ration 4. The fur coats of the animals fed the laboratory chow diets appeared more glossy and better in texture than the fur of those fed ration 4.

Gross Appearance of Periodontal Lesions.—The periodontal lesions in the soft tissues of the hamsters were characterized by food impaction in the interproximal regions and by recession and detachment of the gingival tissue from the tooth surface. In some specimens, marginal gingivitis accompanied by deep pockets that often were filled with food debris was observed (Fig. 1).

Their were no hair impactions in the periodontal pockets, and rarely was salivary calculus observed. The periodontally involved teeth showed slight to moderate mobility. After removal of the soft tissue, areas of bone resorption were observed (Fig. 2). The degree of bone resorption appeared to be related to the amount of food impaction.

Evaluation of Periodontal Lesions.—The average number of periodontal lesions and the average extent of these lesions are shown in Table I. From the data presented, it can be seen that the desalivated animals maintained on the three diets exhibited a higher number and greater extent of both the soft- and calcified-tissue lesions than their intact controls. Highly statistically significant differences between the control and desalivated animals maintained on either ration 4 or laboratory chow were observed. Desalivated animals maintained on ground chow showed little or no significant increments in the soft-tissue lesions; however, the increments in the calcified-tissue lesions of the

TABLE I. THE EFFECTS OF DESALIVATION UPON THE INCIDENCE OF PERIODONTAL LESIONS IN THE SYRIAN HAMSTER*

NO.	GROUPS	NO. OF ANIMALS	NO. OF WEEKS	SOFT-TISSUE LESIONS				CALCIFIED-TISSUE LESIONS			
				NUMBER OF PERIODONTAL LESIONS		EXTENT OF PERIODONTAL LESIONS		NUMBER OF PERIODONTAL LESIONS		EXTENT OF PERIODONTAL LESIONS	
				AVERAGE	C.R.†	AVERAGE	C.R.	AVERAGE	C.R.	AVERAGE	C.R.
1	Control (ration 4)	12	12	22.1 (1.6)	3.3	34.4 (4.9)	3.3	24.4 (1.0)	3.0	40.0 (2.0)	5.3
2	Desalivated (ration 4)	10	12	27.8 (0.6)		68.5 (9.2)		27.8 (0.6)		69.8 (5.2)	
3	Control (laboratory chow)	10	12	15.0 (1.4)	3.0	15.6 (1.5)	3.7	21.6 (1.1)	4.0	31.1 (1.7)	4.2
4	Desalivated (laboratory chow)	9	12	22.3 (2.2)		34.3 (4.9)		26.0 (0.3)		44.6 (2.7)	
5	Control (ground chow)	11	12	20.7 (1.3)	2.0	24.9 (2.3)	1.3	22.2 (1.6)	3.6	32.1 (2.3)	4.0
6	Desalivated (ground chow)	8	12	24.0 (1.0)		33.0 (5.6)		28.0 (0.0)		55.7 (5.5)	

*The value within parentheses represents the standard error of the mean.

†C.R. = Critical ratio, the ratio of the difference between two means to the standard error of the difference between the means. Wherever the critical ratio is less than 2.0, the difference between the means is considered to be statistically insignificant; when the critical ratio is between 2.0 and 2.9, the difference is of borderline significance; when the ratio is 3.0 or higher, the difference is highly significant.

TABLE II. THE INCIDENCE OF PERIODONTAL LESIONS ASSOCIATED WITH DIFFERENT DIETS*

GROUP	RATION	SOFT-TISSUE LESIONS				CALCIFIED-TISSUE LESIONS			
		NUMBER OF PERIODONTAL LESIONS		EXTENT OF PERIODONTAL LESIONS		NUMBER OF PERIODONTAL LESIONS		EXTENT OF PERIODONTAL LESIONS	
		AVERAGE	C.R.†	AVERAGE	C.R.	AVERAGE	C.R.	AVERAGE	C.R.
1	Ration 4	22.1 (1.6)	3.4	34.4 (4.9)	3.6	24.4 (1.0)	2.0	40.0 (2.0)	3.4
2	Laboratory chow	15.0 (1.4)	0.7	15.6 (1.5)	1.7	21.6 (1.1)	1.3	31.1 (1.7)	2.6
3	Ground laboratory chow	20.7 (1.3)	3.0	24.9 (2.3)	3.5	22.2 (1.6)	0.3	32.1 (2.3)	0.1

*The value in parentheses represents the standard error of the mean.
†C.R. = Critical ratio.

desalivated animals were of high statistical significance. These data indicated that the incidence of soft- and calcified-tissue lesions increased upon removal of the major salivary glands in Syrian hamsters.

The incidence of periodontal lesions in the control (intact) animals maintained on three diets of varying consistency and composition is presented in Table II. The control animals on ration 4 had the maximum number and extent of soft-tissue lesions, while the control animals on the laboratory chow pellets showed the minimum number and extent of soft-tissue lesions. The differences for the two groups were of high statistical significance. The animals on ground chow had fewer and less extensive soft-tissue lesions than those on ration 4,

Fig. 1.

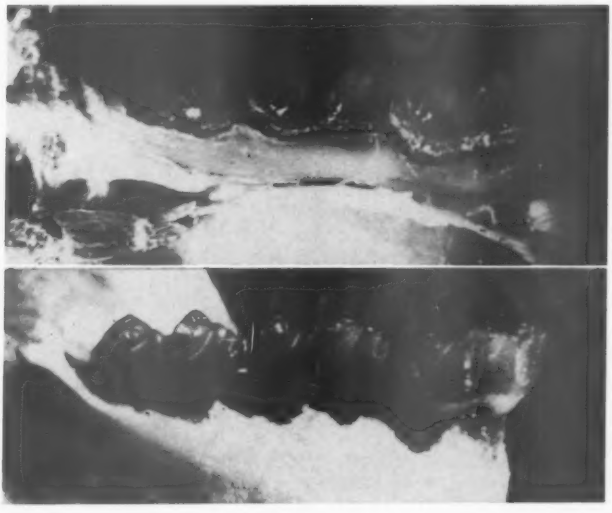


Fig. 2.

Fig. 1.—Soft-tissue lesions in the Syrian hamster.
Fig. 2.—Calcified-tissue lesions of the specimen shown in Fig. 1, after removal of the soft tissues.

but the differences were not statistically significant. The number and extent of soft-tissue lesions in animals fed laboratory chow pellets were significantly lower than those of the animals fed ground laboratory chow. These data suggested that ration 4 produced severe soft-tissue lesions, while laboratory chow pellets induced relatively mild lesions. The laboratory chow, when ground, produced relatively severe soft-tissue lesions.

Somewhat comparable findings were observed for the calcified-tissue lesions. Animals fed ration 4 exhibited the maximum number and extent of calcified-tissue lesions, while the animals on laboratory chow pellets showed a minimal number and extent of such lesions. The differences for the two groups were of moderate statistical significance for the number of lesions

but of high statistical significance for the extent of lesions. The number and extent of lesions produced by ground laboratory chow were fewer than those produced by ration 4. The differences were not statistically significant for the number of lesions however, and they were of only moderate statistical significance for the extent of lesions. No statistically significant differences were found in the number and extent of calcified-tissue lesions produced by laboratory chow pellets and ground laboratory chow. These data indicate that animals on ration 4 showed a greater number and extent of calcified-tissue lesions than those fed either laboratory chow or ground laboratory chow. In addition, the three diets showed more severe periodontal involvement in the soft tissues than in the calcified tissues.

HISTOLOGIC FINDINGS

For the purpose of this study, the periodontiums of the first and second right maxillary molars were used as representative specimens.

Animals Maintained on Ration 4.—

Control group: The epithelium of the gingival papilla appeared somewhat edematous. The underlying connective tissue exhibited a mild round-cell infiltration limited to the transseptal fiber level. Some edema in the gingival fibers was also present. The mild inflammatory changes noted in these animals were always observed in association with food impaction. The epithelial attachment was at the cementoenamel junction (Fig. 3).

The fiber bundles of the periodontal membrane showed normal functional orientation. Bone formation was observed at the crest, as evidenced by the presence of osteoid at the surface. Osteoblasts were noted to line the alveolar crest (Fig. 3). Enlargement of the marrow spaces of the interdental and interradicular septa was frequently noted.

Desalivated group: The changes in the epithelium were essentially similar to those found in the control animals, except that these changes were of greater severity. Ulceration of the interproximal gingival epithelium associated with severe food impaction was noted (Fig. 4). The presence of inflammatory cellular elements was observed in the subepithelial connective tissue. Apical migration of the epithelial attachment was noted.

A prominent observation was the presence of epithelium in the bifurcation of the roots (Fig. 5). Active bone resorption characterized by deep lacunae containing osteoclasts was observed at the crestal area. The marrow spaces were markedly enlarged (Fig. 6).

Animals Maintained on Laboratory Chow.—

Control group: A slight inflammatory infiltrate was noted subepithelially at the gingival papilla. No other abnormalities were noted in the periodontal structures of this group (Figs. 7 and 8).

Desalivated group: The gingival papillae showed some edema and an inflammatory infiltrate in the subepithelial connective tissue (Fig. 9).

Fig. 3.

Fig. 4.

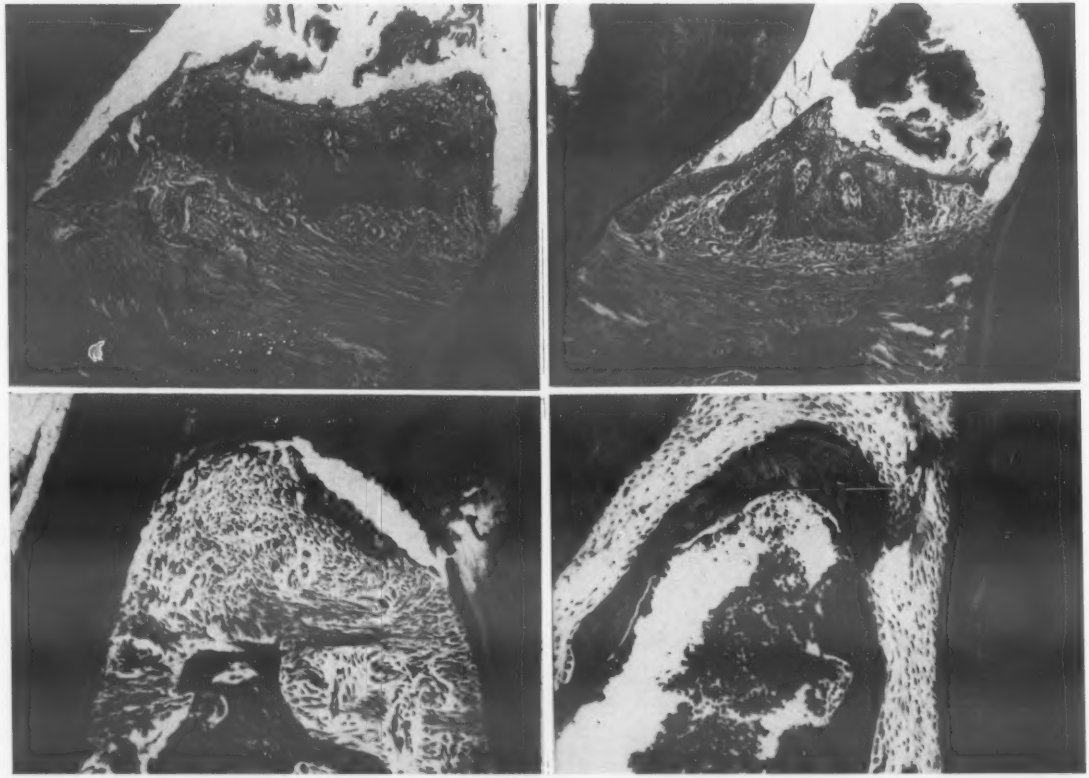


Fig. 5.

Fig. 6.

Fig. 3.—Photomicrograph of an interproximal papilla between the first and second maxillary molars in a control hamster fed ration 4. Note the epithelial attachment at the cementoenamel junction. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 4.—Photomicrograph of an interproximal area between first and second maxillary molars in a desalivated hamster fed ration 4. Note the apical migration of the epithelial attachment. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 5.—Photomicrograph of an interradicular area of a maxillary first molar in a desalivated hamster fed ration 4. Note the presence of epithelium at the cementum of the interradicular crest. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 6.—Photomicrograph of an interradicular area of a maxillary first molar in a desalivated hamster fed ration 4. Note extensive osteoporosis of the interradicular septum. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 7.

Fig. 8.

ence of epithelium at the cementum of the interradicular crest. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 6.—Photomicrograph of an interradicular area of a maxillary first molar in a desalivated hamster fed ration 4. Note extensive osteoporosis of the interradicular septum. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 7.

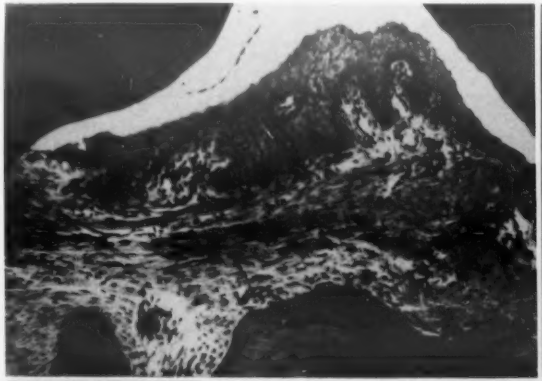
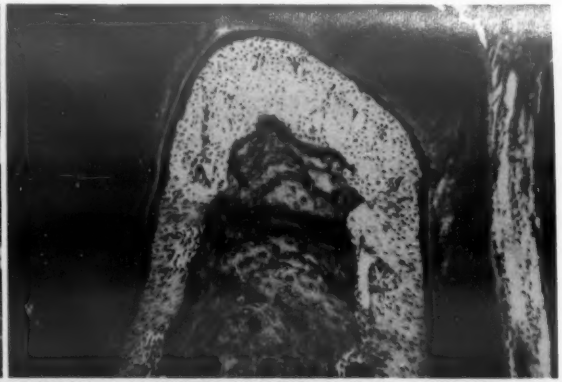
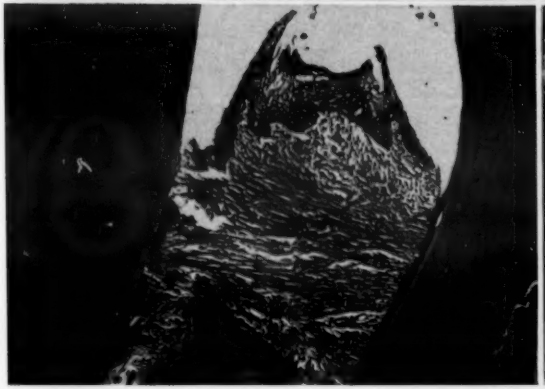


Fig. 8.





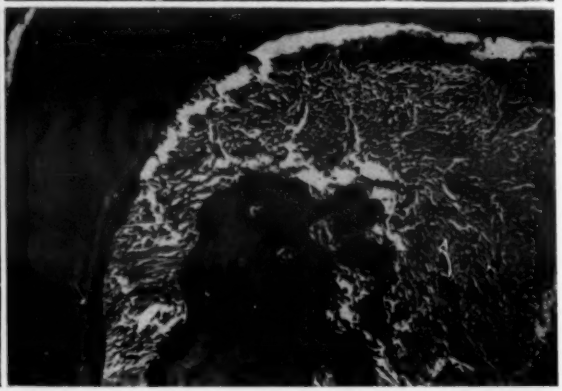


Fig. 9.

Fig. 10.

Fig. 7.—Photomicrograph of an interproximal area between first and second maxillary molars in a control hamster fed laboratory chow. Note slight inflammatory infiltrate in the subepithelial connective tissue. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 8.—Photomicrograph of an interradicular area of a maxillary first molar in a control hamster fed laboratory chow. Note the normal appearance of the crestal tissues. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 9.—Photomicrograph of an interproximal area between first and second maxillary molars of a desalivated hamster fed laboratory chow. Note edema in the subepithelial connective tissue. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 10.—Photomicrograph of an interradicular area of a maxillary first molar in a desalivated hamster fed laboratory chow. Note the presence of epithelium in the bifurcation and slight crestal bone resorption. (The space between cementum and dentine is an artifact.) (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

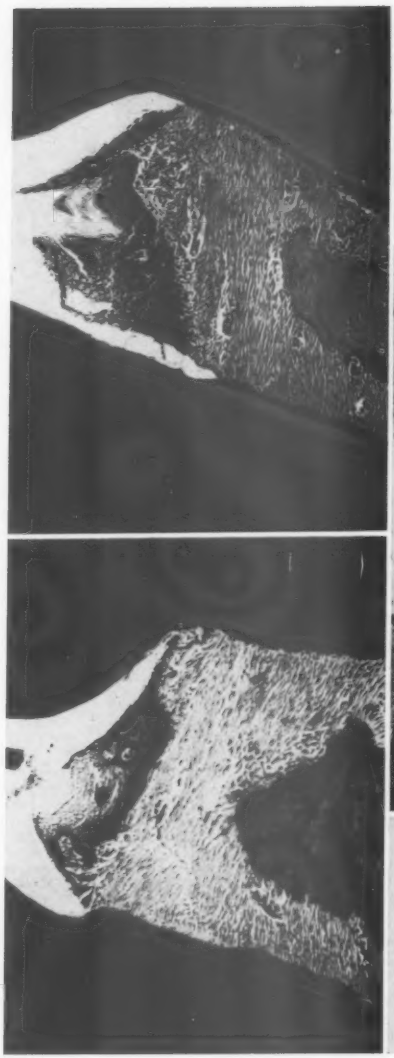


Fig. 11.

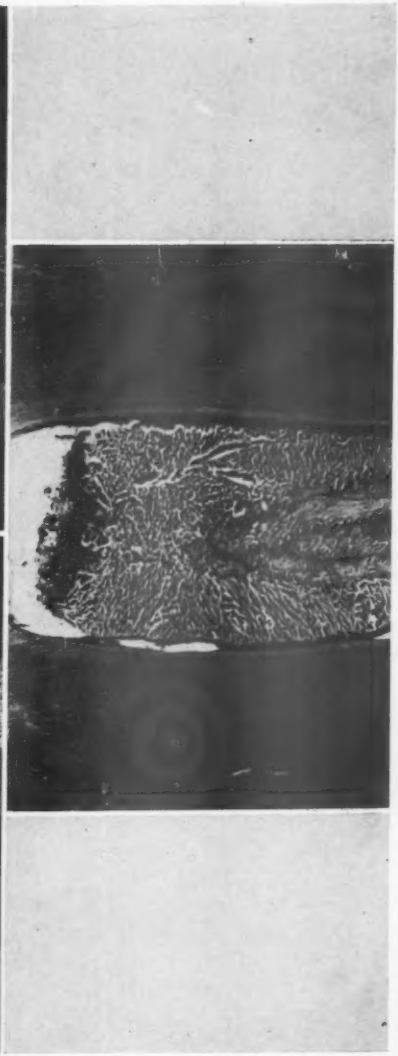


Fig. 12.

Fig. 13.

(For legends, see opposite page.)

The periodontal membrane fibers were normally oriented. The presence of epithelium at the interradicular area was prominent. Slight bone resorption at the interdental and interradicular crests was observed (Figs. 9 and 10).

Animals Maintained on Ground Laboratory Chow.—

Control group: The gingival papilla showed some inflammatory cells below the epithelium which was often associated with food impaction (Fig. 11). The alveolar bone showed normal functional adaptations.

Desalivated group: The gingival papilla showed slight apical proliferation of the epithelial attachment with evidence of inflammation (Fig. 12).

The functional arrangement of the periodonal membrane fibers was noted. The presence of epithelium below the root bifurcation was prominent (Fig. 13).

DISCUSSION

The results of these studies suggest that the incidence of periodontal lesions in the soft and calcified tissues of the hamsters increases after removal of the major salivary glands. The above findings were observed both macroscopically and microscopically. These findings represent the response of the periodontal tissues to the altered local and systemic environment. After removal of the salivary glands, the production of periodontal lesions may have been influenced in the following ways: (1) the lack of saliva could enhance the accumulation of food around the teeth and soft tissues, (2) there may be some unknown factor in saliva which is essential to the integrity of the soft tissues, and (3) the periodontal changes may be due to the combined effects of the preceding two factors. From the present investigation, it is not possible to determine which of the above factors is the cause of periodontal lesions in these animals. However, further studies will throw light upon this problem.

The consistency of the diet appeared to influence the production of periodontal lesions in the hamster. A finely powdered, high-carbohydrate, cariogenic ration 4 produced periodontal lesions of maximum severity, while laboratory chow in pellet form produced relatively mild periodontal lesions. When the laboratory chow pellets were ground to a moderately fine particle-sized powder, however, the periodontal destruction was increased. These findings are in sharp contrast to the findings of Auskaps, Gupta, and Shaw[9] but are similar to those of Stahl, Miller, and Goldsmith.[10] On the basis of these studies, the influence of the particle size of the diet definitely must be given consideration.

Fig. 11.—Photomicrograph on an interproximal area between the first and second maxillary molars in a control hamster fed ground laboratory chow. Note epithelial attachment at the cementoenamel junction and evidence of slight inflammatory cells. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 12.—Photomicrograph of an interproximal area between the first and second maxillary molars of a desalivated hamster fed ground laboratory chow. Note slight apical migration of epithelial attachment. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

Fig. 13.—Photomicrograph of an interradicular area of a maxillary first molar in a desalivated hamster fed ground laboratory chow. Note the presence of epithelium at the bifurcation and active osteoclastic activity at the crest. (Hematoxylin and eosin stain. Magnification, ×100; reduced ¼.)

From the present studies, it is apparent that when destructive changes occurred in the soft and calcified tissues, their severity was accentuated by removal of the major salivary glands. The severity of periodontal involvement seemed to be dependent upon the existing preoperative periodontal lesion. It will be interesting to explore the effects of sialoadenectomy in animals in which periodontal lesions cannot ordinarily be produced.

The consistent histopathologic finding of epithelium at the interradicular area in the desalivated animals shows the severity of periodontal involvement. The observations of osteoporosis characterized by widening of the marrow spaces in the alveolar bones of both the control and the desalivated animals maintained on ration 4 are similar to those produced by protein deprivation[11, 12] and by acute starvation.[13] This raises the question as to whether ration 4 is nutritionally adequate.

SUMMARY

Sixty hamsters were used to study the effects of desalivation on the incidence of periodontal lesions under three different dietary regimens.

The desalivated animals consistently showed a greater number and extent of periodontal lesions in the soft and calcified tissues than their controls. Highly statistically significant differences were observed between the lesions of the control animals and those of the desalivated animals.

The consistency and composition of diet seemed to play a role in the production of periodontal lesions in the hamster. High-carbohydrate, finely powdered, cariogenic ration 4 produced a greater number and extent of soft- and calcified-tissue lesions than either the laboratory chow or the ground laboratory chow. Ground laboratory chow produced a greater number and extent of lesions than laboratory chow in pellet form. The effects of the diets on the soft tissues appeared to be more severe than on the calicified tissues.

The microscopic findings substantiated the gross observations. The desalivated animals showed histologic changes of greater severity than the intact controls. The pronounced changes in the desalivated animals were apical migration of the epithelial attachment and the presence of epithelium at the interradicular area. The control and desalivated animals fed ration 4 showed severe osteoporosis, possibly due to the nutritional inadequacy of the diet.

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STUDIES IN DIAGNOSIS IN ORAL SURGERY AND ORAL MEDICINE

Lester R. Cahn and Kurt H. Thoma

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HYPERPARATHYROIDISM FIRST SUSPECTED THROUGH A GIANT-CELL EPULIS

Report of a Case

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INTRODUCTION

HYPERPARATHYROIDISM, a rare and complex endocrine disturbance, is usually caused by an adenoma of a parathyroid gland and is characterized clinically by renal, skeletal, or gastrointestinal symptoms. The diagnosis depends upon abnormal chemical findings in the blood and urine. The finding of "giant-cell epulis" as the presenting symptom of hyperparathyroidism is rare and has prompted this report. Because of the importance of early diagnosis and treatment if irreversible kidney damage is to be prevented, a brief review of the clinical and laboratory features of hyperparathyroidism and of giant-cell lesions of the oral cavity will be presented.

GIANT-CELL LESIONS OF THE ORAL CAVITY

The name "giant-cell lesion" may be assigned to at least three oral histopathologic entities: (1) the giant-cell tumor, a true neoplastic lesion; (2) the giant-cell reparative granuloma, a reparative type of lesion; and (3) the giant-cell lesion that represents one of the oral manifestations of hyperparathyroidism.

Giant-Cell Tumor.—The giant-cell tumor supposedly occurs in patients beyond the age of 20, but Waldron[14] states that most of his cases were in children 7 to 11 years old. This tumor develops in bones that are of cartilaginous origin. In the mandible, the condyle and premolar region are frequent sites.

From the Head and Neck Surgery Section, Department of Surgery, and the Oral Surgery Section, Department of Dentistry, Long Island Jewish Hospital.

The entire maxilla, particularly the canine area, may be involved. The bony plates are usually expanded and the adjacent teeth are separated by the tumor which, on gross examination, is brownish in color. The x-ray picture of a giant-cell tumor usually shows extensive irregular destruction of the bone. Histopathologically, the tumor reveals an invasive character. The giant cells are abundant, large, and intermixed with spindle and round cells. The fibrous stroma shows cytoplasmic and nuclear irregularities. Because of the possible malignant features, the tumor must be removed adequately. Curettage, which is usually adequate for a giant-cell reparative granuloma, is contraindicated in the treatment of this tumor. A wide and adequate resection of the area involved is necessary to prevent recurrence.

Giant-Cell Reparative Granuloma.—The giant-cell reparative granuloma may occur as either a peripheral (extraosseous) or a central (intraosseous) granuloma. According to Jaffe,[5] the lesion occurs primarily in patients between the ages of 10 and 25 years. It is frequently associated with the removal of teeth and other forms of traumatic injuries. The reparative granuloma is a vascular, firm, purplish lesion. It grows fairly rapidly and usually is painless. It must be differentiated from the more serious giant-cell tumor. The histopathologic appearance of a giant-cell granuloma is characterized by a central mass of giant cells in a fibroendothelial stroma. The surface of the peripheral granuloma is usually covered with normal stratified squamous epithelium.[12]

The extraosseous reparative granuloma must be removed with a wide base in order to include the pedicle. The teeth adjacent to the mass may frequently require removal to ensure adequate accessibility and visibility to the base of the lesion. The central giant-cell granuloma should be thoroughly curetted. The prognosis is excellent if the curettage removes the entire lesion.[13]

Giant-Cell Lesion of Hyperparathyroidism.—The dentist may detect the first manifestations of hyperparathyroidism. This may appear as an osteolytic lesion of the mandible or maxilla or as a peripheral lesion. The giant-cell lesion of hyperparathyroidism cannot be differentiated histologically from the giant-cell reparative granuloma. Grossly, the blood pigment imparts a brownish color to the tumor; hence, the name "brown tumor of hyperparathyroidism." The bony changes in the maxilla and mandible may appear as loss of the lamina dura, loss of trabecular structure, or multiple cystlike lesions.

HYPERPARATHYROIDISM

Clinical Picture.—The disease is chronic and slowly progressive. The symptomatology is quite varied, resulting in delayed diagnosis and treatment. Hyperparathyroidism may occur at any age after puberty, and it is reported as being three times as frequent in females as in males. In Rienhoff's[10] experience, renal complications were more frequent in males, whereas osseous manifestations were more common in females. The average duration of symptoms prior to the discovery of the adenoma ranged from five to seven years, which emphasizes the insidious course of the disease. The parathyroid adenoma is usually small, and mechanical symptoms due to its presence are rare. In

fact, in most reported series, the adenoma is palpable in only 10 to 20 per cent of the cases. Most of the symptoms are due to the hypercalcemia which is caused by the excessive secretion of parathormone. There is a decreased response of nerves and muscles, resulting in general muscular atony. Lassitude, easy fatigability, weakness, and lethargy are frequent. Anorexia, nausea, and vomiting are often present, and sometimes there is severe abdominal pain.[9] Rogers[11] has focused attention on the association of duodenal ulcer and hyperparathyroidism, and Cope has found an ulcer in nearly one-fifth of his cases. All the above symptoms are nonspecific, and it is for this reason that the malady is not recognizable, except in rare instances, until the complications become evident.

The complications of hyperparathyroidism usually involve the kidneys and the skeletal system and are so prominent that they have often been regarded as the disease itself. Excessive excretion of calcium and phosphorus in the urine favors the formation of calculi in the renal pelvis or deposition of calcium in the renal tubules. It should be emphasized that a mild degree of hyperparathyroidism may result in sufficient stone formation to kill the patient. Symptoms due to skeletal involvement vary from vague aches and pains to the extremely deforming osteitis fibrosa cystica. In some cases there is generalized demineralization, while in others localized cysts or tumors occur. Pathologic fractures are frequent. In extreme cases, kyphosis, loss of height, and other deformities are seen.

The skeletal aspect of the disease was recognized long before the renal aspect.[2] As more cases of renal calculus are being studied chemically, it is becoming increasingly apparent that urinary tract complications of hyperparathyroidism occur earlier and with greater frequency than the osseous changes.[7] It has been estimated that in about 5 per cent of the patients with renal calculi parathyroid overactivity causes the stones.

Laboratory Data.—The preoperative diagnosis of hyperparathyroidism depends upon the laboratory demonstration of a disturbance of calcium and phosphorus metabolism. The chief criteria are (1) hypercalcemia, (2) hypophosphatemia, (3) hypercalciuria, and (4) elevated serum alkaline phosphatase.

The normal range of serum calcium is 8.5 to 10.2 mg. per 100 c.c. In hyperparathyroidism the level is elevated, often above 12 mg. per 100 c.c. Some cases show only slight elevations above the normal. The values are usually higher in the cases of classic bone disease than in those with minimal or no bone changes. The serum inorganic phosphorus varies normally from 3.0 to 4.0 mg. per 100 c.c. The amount of depression of the phosphate level in hyperparathyroidism may be slight and may vary with repeated determinations, but this change is more constantly present than is hypercalcemia, according to Albright.[1] In the presence of renal insufficiency, the depression of phosphorus may be obscured by the tendency toward phosphorus retention and the serum phosphate level may be normal or minimally elevated.

The quantitative determination of twenty-four-hour excretion of calcium in the urine in patients maintained on a low-calcium diet for several days is an accurate and valuable adjunct to the diagnosis of hyperparathyroidism. A

normal person excretes less than 100 mg. of calcium daily, while values above 150 mg. are highly suspicious and those above 200 mg. are definitely diagnostic of the disease. This examination is rather difficult for routine use, and the qualitative test devised by Sulkowitch provides a rapid means of estimating urinary calcium excretion.

The normal alkaline phosphatase value is 4 to 10 King-Armstrong units per 100 c.c. There is usually an elevation of the alkaline phosphatase in cases with bone disease, while the test is normal in patients with no skeletal involvement. The level of circulating alkaline phosphatase is dependent in part on the activity of the osteoblasts, which increases as a result of the decalcification in hyperparathyroidism. It is thus a reflection of bone change rather than of hyperparathyroidism per se.

Early cases of hyperparathyroidism with minimal or unusual clinical manifestations may have minimal hypercalcemia and normal phosphorus values in the blood, especially if the patients are on high-phosphorus intake. Two diagnostic tests recently introduced have been extremely useful in the diagnosis of these cases. These are the tubular reabsorption of phosphorus[3] and the calcium tolerance test.[8]

Pathogenesis.—In nearly all cases hyperparathyroidism is caused by an adenoma of the parathyroid gland. Slightly less than 10 per cent of cases are due to diffuse hyperplasia of all the parathyroid glands, and in rare instances the disease is due to carcinoma of a parathyroid gland. The location of normal as well as abnormal parathyroid glands is extremely variable, glands having been found from 1 cm. above the upper pole of the thyroid gland down to the level of the pericardium in the mediastinum. The size of the adenomas may also vary widely, from a few millimeters to several centimeters in size. Experience in the identification of normal parathyroid glands is essential for the surgeon exploring a patient with the diagnosis of hyperparathyroidism.

The manifestations of the disease are due to the increased production of parathormone by the adenomas. The hormone, which is not dependent on the pituitary for its secretions,[6] is believed to act primarily on the renal tubules, causing a decrease in the reabsorption of phosphorus.[4] With a loss of serum phosphorus, an imbalance is created between the calcium and phosphorus ions in the blood. In an attempt to restore the ionic balance, calcium is withdrawn from the skeletal structures, with a resultant hypercalcemia and subsequent demineralization of the bones. However, a high oral intake of calcium will minimize the bony changes.

Treatment.—Once the diagnosis of hyperparathyroidism is made, surgical exploration is mandatory in order to locate and remove the parathyroid adenoma. This may be a relatively simple operation when the tumor is located in the region of the thyroid gland, or it may be exceedingly difficult to perform if the adenoma is located in the mediastinum or embedded in the thyroid gland itself, or if the disease is not due to an adenoma but to hyperplasia of all four parathyroid glands. At operation a thorough search of the entire anterior cervical region must be made. It is essential to identify all four parathyroid glands, even though a tumor is discovered, because of the possibility of multiple

adenomas. If no tumor is found, and if there is no diffuse hyperplasia of all four parathyroid glands, the search must be extended into the mediastinum. The tumor or tumors must be removed completely and intact, because of the possibility of carcinoma. If diffuse hyperplasia of the four glands is found, three of the glands and a major portion of the fourth gland must be resected to effect a cure.

The type of postoperative course depends to a large extent upon the clinical picture presented by the patient. Cases with marked skeletal involvement will develop severe reduction of serum calcium and phosphorus because of the rapid postoperative deposition of calcium in the demineralized bones. Tetany will ensue unless massive doses of calcium without phosphate are administered. Patients who have little or no bony changes preoperatively usually show no significant hypocalcemia after operation. It is best to keep bone remineralization at a slow rate in order to prevent tetany. This can be done by restricting foods with high phosphorus content, such as milk and dairy products. Calcium may be given in the form of lactate or gluconate. Dihydrotachysterol (A.T. 10) or large doses of vitamin D may be used to increase calcium absorption. An excellent guide in the postoperative course is the urinary Sulkowitch test; a rapid fall to normal values is usual after successful surgery. If the Sulkowitch test drops to zero, blood studies are indicated to exclude tetany.

The degree of recovery of renal function is dependent upon the amount of damage which has occurred preoperatively. In general, the prognosis in cases of hyperparathyroidism depends upon the successful removal of the adenoma or adenomas and upon the degree of irreversible changes that have occurred in the kidneys due to the deposition of calcium.

CASE REPORT

M. G., a 44-year-old white male clerk, was admitted to the Long Island Jewish Hospital on March 23, 1958. Four months prior to admission he had a left lower second molar extracted because of extensive caries. Following the extraction, the patient was noted to have a mass in the dental socket, and he was referred to an oral surgeon. Clinical examination at that time revealed a soft, bluish, irregular, friable mass measuring approximately 2 by 1 cm., which involved the entire lower left molar area, extending from the buccal area to the lingual area and from the socket to the occlusal level of the adjacent teeth (Fig. 1).

Family History.—The patient's father died of heart disease and one brother had diabetes mellitus.

Past History.—During World War II he had received a bullet wound in the right brachial plexus. He had undergone psychiatric treatment in the past for "anxiety neurosis." Systemic review disclosed recent easy fatigability, cramps in the legs, polyuria with some burning on urination, and abdominal cramps associated with heartburn. A gastrointestinal study about one year previously revealed a "healing peptic ulcer."

Dental X-ray Examinations.—Roentgenograms of the second molar area prior to the extraction showed a solitary, small, radiolucent area adjacent to the mesial apex of the lower left second molar (Fig. 2). There was a diffuse radiolucency in the lower left second premolar—first molar region. X-ray examination four months later showed a similar appearance in the molar region; the bony structure adjacent to the remainder of the mandibular teeth was essentially negative (Fig. 3).

Operative Procedure.—The patient was operated upon under local anesthesia supplemented by nitrous oxide-oxygen analgesia. The mass in the left lower molar region was excised

Fig. 1.—Gross appearance of soft-tissue mass in lower left molar region.

Fig. 2.—Preoperative roentgenogram showing lower left molar in position with bony changes mesial to the apices.

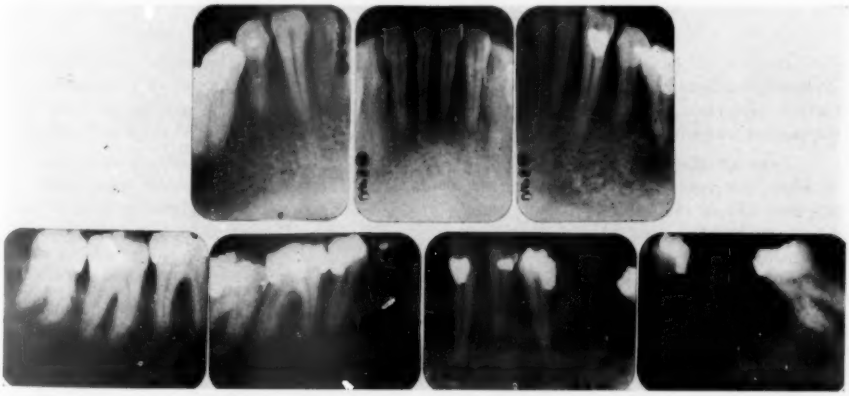

Fig. 3.—X-ray series of mandibular teeth showing bony changes in lower left molar region.

with a border of normal tissue from the buccal and lingual gingivae. It was then separated from the adjacent bone with difficulty. The defect was sutured to effect hemostasis.

Pathologic Report.—

Gross description: The specimen was an irregular piece of tissue, 2 by 1.5 by 1 cm. in size. One surface was partially bordered by a crescentic segment of pale gray, smooth tissue. The remainder showed a heaped-up, granular, reddish brown appearance. On section, the underlying material had a hemorrhagic, reddish brown discoloration.

Microscopic description: Sections revealed a squamous mucosa which was ulcerated and which contained a marked acute inflammatory reaction in the base of the ulcer. Underlying the mucosa, there was a mass of tissue composed of numerous giant cells in a very vascular fibrous tissue stroma. In addition to the giant cells, there was a liberal intermixture of plasma cells and lymphocytes (Fig. 4).

Diagnosis: Giant-cell epulis.

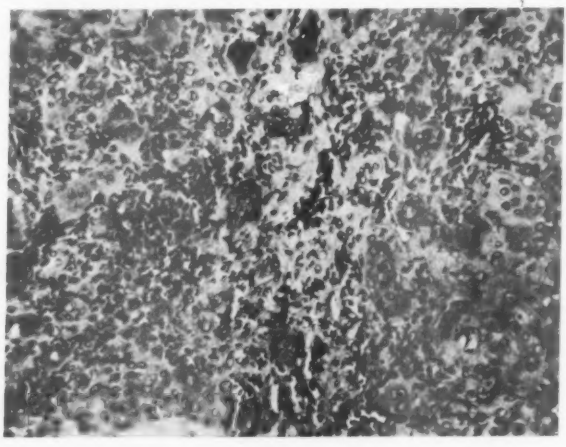

Fig. 4.—Photomicrograph of lesion seen in Fig. 1, showing numerous giant cells. (Magnification, ×225; reduced ¼.)

Because of the pathologic report and the x-ray findings in the mandible, the possibility of hyperparathyroidism was entertained and the patient was referred to an internist for further evaluation. Laboratory studies revealed hypercalcemia and hypophosphatemia, and the patient was admitted to the Long Island Jewish Hospital for further study and treatment.

Physical Examination.—The patient was a well-developed and well-nourished man in no acute distress. Upon examination of the neck, a soft, ill-defined, ovoid mass could be palpated low in the right peritracheal region, which moved up with deglutition. There was a healed scar in the region of the right shoulder, and there was some atrophy of the intrinsic muscles of the right hand with decreased sensation along the distribution of the right ulnar nerve. The remainder of the physical examination was essentially negative.

Laboratory Findings.—The hemoglobin was 13.2 Gm., and the white blood count was 6,550 with a normal differential. The hematocrit was 42. Urinalysis revealed yellow, clear, acid urine with a specific gravity of 1.009 and occasional white cells; it was negative for albumin, sugar, and acetone. Urine Sulkowitch (qualitative test for urinary calcium) was 2-plus on two occasions. Other findings included serum calcium, 12.7 mg. per 100 c.c.; serum phosphorus, 1.5 mg. per 100 c.c.; blood urea nitrogen, 18 mg. per 100 c.c.; plasma proteins, 6.3 Gm. per 100 c.c.; and alkaline phosphatase, 30 King-Armstrong units. The patient was placed on a Bauer-Aub (calcium-free) diet upon admission to the hospital, and four days

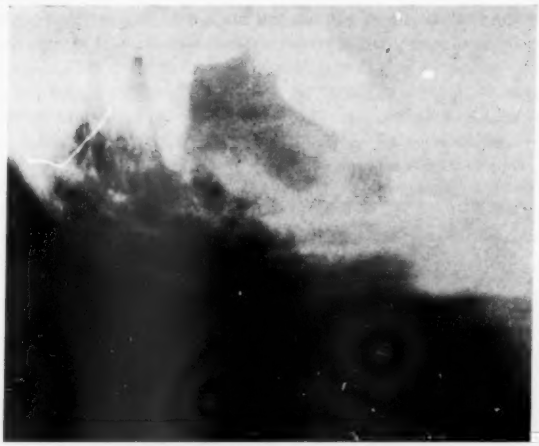

Fig. 5.—Lateral roentgenogram of left mandible with cystic changes in the region of the left premolars and canines.

Fig. 6.—Roentgenogram of the hand showing the typical subperiosteal resorption of the phalanges.

later quantitative urine calcium and phosphorus studies were performed. These revealed a twenty-four-hour volume of 1820 c.c. of urine, with a calcium of 504 mg. and phosphorus of 91 mg. in 24 hours.

Roentgenologic Findings.—Examination of both mandibles showed a diffuse granular osteoporosis. The lamina dura was absent. Some cystic changes were seen in the region of the left premolars and canines (Fig. 5). Roentgenograms of the skull revealed that the cranial vault was demineralized and presented a granular appearance. The hands showed a considerable degree of demineralization of all the bones of the hands and wrists, with typical subperiosteal resorption of the phalanges (Fig. 6). The long bones showed generalized demineralization with lytic changes in the proximal femurs. A small indentation was seen on the right side of the trachea at the level of the thoracic inlet.

Fig. 7.—Gross appearance of parathyroid tumor removed at operation.

Diagnosis.—The case was diagnosed as primary hyperparathyroidism due to parathyroid adenoma, probably located in the right cervical region.

Operative Procedure.—On March 28, 1958, after induction of general endotracheal anesthesia, a collar incision was made in the lower anterior cervical region and carried down through the skin, fat, and platysma. The skin flaps were elevated up to the level of the thyroid notch and down to the sternum. The deep cervical fascia was incised in the midline, and the strap muscles were retracted laterally to expose the thyroid gland, which was found to be entirely normal in appearance. The right side was explored first. After the inferior and middle thyroid veins had been ligated and divided, the right lobe of the thyroid gland

was lifted out of its bed and delivered into the wound and the parathyroid tumor immediately came into view. It appeared as an ovoid, well-encapsulated, lobulated, soft mass measuring 5 by 2.5 by 1.5 cm. It lay superficial to the recurrent laryngeal nerve. It was brownish in color, in contrast to the adjacent pink thyroid gland. The tumor was removed intact after ligation of its vascular pedicle. The inferior parathyroid on the right side and two normal parathyroid glands on the left side were then identified and preserved intact. The wound was closed in layers.

Fig. 8.—Microscopic appearance of parathyroid adenoma showing cords of chief cells and clear cells. (Magnification, ×230; reduced ¼.)

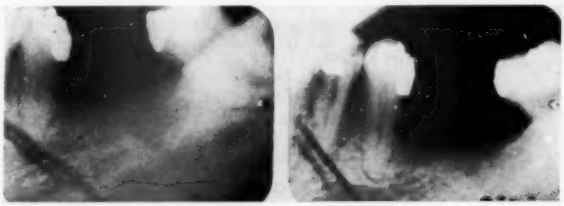

Fig. 9.—Roentgenogram of left molar area six months postoperatively showing recalcification of the bone surrounding the molars.

Pathologic Report.—

Gross: The specimen consisted of a nodule measuring 5 by 2.5 by 1.5 cm. and weighing 14 grams. The capsule was intact, but on palpation the mass was felt to be nodular in consistency. On sectioning, the lesion was found to be extremely hemorrhagic. It consisted generally of red translucent tissue, however, except at one pole where there was a nodular, fleshy, pink-white area (Fig. 7).

Microscopic: The tumor consisted of anastomosing cords of chief cells and clear cells. It was extremely vascular to the point where dilated thin-walled and thick-walled vascular channels were present in such a large number as to appear almost to represent a vascular malformation. Examination of the individual cells comprising the tumor did not reveal mitotic figures. The capsule was noted to be intact, and no evidence of invasion into the capsule was seen (Fig. 8).

Pathologic diagnosis: Parathyroid adenoma.

Postoperative Course.—The patient convalesced satisfactorily. The only complaint was some tightness and soreness in the muscles of the lower extremities. There was no clinical evidence of tetany. On the second postoperative day the serum calcium was 10.7 mg. per cent and serum phosphorus was 3.5 mg. per cent. Quantitative urinary excretion was studied again on March 31 and revealed a twenty-four-hour volume of 1,160 c.c., with a calcium of 243 mg. and phosphorus of 55 mg. in twenty-four hours. The patient was re-examined in September, 1958, and again in December, 1958, and was completely asymptomatic. He no longer experienced weakness and easy fatigability, and he had no gastrointestinal complaints. There had been complete healing of the gingivae, and x-rays revealed recalcification of the bones in the vicinity of the molar teeth (Fig. 9).

SUMMARY

1. We have reported a case of primary hyperparathyroidism in which the first clinical suspicion was aroused by the finding of a giant-cell lesion in the oral cavity.

2. Giant-cell lesions of the oral cavity are reviewed and differentiated.

3. The clinical picture, laboratory findings, pathogenesis, and treatment of hyperparathyroidism are briefly discussed.

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DISCUSSION BY DR. LESTER R. CAHN

Hyperparathyroidism today is not considered as rare a disease as it was not too many years ago. As more and more cases, exhibiting varied symptoms, have appeared in the literature and as more precise biochemical techniques have evolved, hyperparathyroidism has become a disease of fairly protean characteristics.

Albright,[1] a number of years ago, showed that bone lesions (that is, the Von Recklinghausen type of hyperparathyroidism) are not as commonly seen in this country as in Europe. He attributed this to the fact that we are great milk consumers, and he showed that calcium could protect, to a large extent, the skeleton from the ravaging effects of an excess of the parathyroid hormone. Therefore, other symptoms have to be taken into account as being suggestive of the malady. Urologists have long been acquainted with the fact that kidney stones and calcific deposits in the urinary tract may be manifestations of hyperparathyroidism. Later such varied symptoms as marked dryness of the throat, gastric disturbances, and mental aberrations were related to the excessive production of parathyroid hormone.

It was formerly thought that children were exempt from the disease. In fact, I have a note from a world authority on bone diseases who says, in discussing the case of a child with a giant-cell lesion of the maxilla, that hyperparathyroidism could be ruled out because the patient was only 11 years of age. In recent years a number of cases of hyperparathyroidism in very young children have been reported. Also, in some cases there appears to be a familial history of the disease. Other endocrine tumors, particularly those of the pancreas and pituitary, have been reported in association with a parathyroid adenoma.

Within just the last year or so Cope and associates reported a new entity in the surgery of hyperparathyroidism—primary chief-cell hyperplasia of the parathyroid glands. They believe that it is in this type of parathyroid lesion that multiple endocrine abnormalities are found. They reported on ten cases. In four of these there were associated tumors of the pancreas and pituitary. In two cases a father and a daughter were afflicted. In two other cases there were jawbone lesions. In one case there was a "cyst" and the laminae durae were absent. In the other, there was a recurrent "cyst" of the mandible.

This report by Attie and Blum is a textbook example of the disease, with the giant-cell lesion of the mandible, the typical biochemical syndrome, and the roentgenologic evidence of the demineralization of the skeleton. The finding of the giant-cell lesion started the investigation. This is as it should be, for it cannot be stated too strongly that no one can distinguish between the giant-cell lesion of hyperparathyroidism and the purely local giant-cell affair. It is most gratifying that the primary mischief was turned up through the diagnostic acumen of the dental practitioner.

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ARMED FORCES AND PUBLIC HEALTH SERVICE

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STUDIES ON A HYDROGENATED FAT-ZINC BACITRACIN PERIODONTAL DRESSING

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In a recent publication, it was shown that a dressing containing 3,000 units of zinc bacitracin per gram of powder is an effective postgingivectomy pack. It is stable at room temperature, easy to use, and affords the patient maximum comfort.[1] Surgical procedures are now being employed, however, which expose fairly extensive areas of alveolar bone. It is not unusual that such surgically exposed areas require one, two, or more weeks for granulation tissue to completely cover exposed bony surfaces. Since this delay in healing might be due, in part at least, to the eugenol content of the dressing, the purpose of this investigation was to develop and clinically test a periodontal dressing which did not contain eugenol.

EXPERIMENTAL

Laboratory Studies.—The formulas for the powder and ointment used to make the periodontal dressing are as follows:

Powder

Zinc oxide	0.45 Gm.
Rosin powder	0.52 Gm.
Zinc bacitracin	3,000 units per gram

Ointment

Zinc oxide	42 per cent
Hydrogenated fat	58 per cent

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The powder is mixed with the ointment until a puttylike consistency is reached. It is then ready for use. If desired, the pack may be mixed weeks ahead of time, wrapped in aluminum foil, and placed in a refrigerator. When needed, it is softened under hot water. If the pack becomes too soft from the hot water, it can be mixed with a little powder until the desired consistency is obtained.

The bacteriostatic effects of the above dressing were studied by comparing it with one which contained both eugenol and sweet almond oil. In both instances the powder portion was the same. Small pellets of each of these freshly prepared dressings were then placed in a Petri dish containing solidified culture medium which had been seeded with fresh saliva before pouring.[1] The plates were incubated at 37° C., and the radius of the zone of inhibition was read and recorded in millimeters at twenty-four- and forty-eight-hour intervals. All tests were run in triplicate.

Findings.—The zones of inhibition around both dressings were the same (4 mm.). This indicated that the bacteriostatic property of the dressing was not lost when the experimental ointment was substituted for the liquid containing eugenol and sweet almond oil.

Clinical Investigation.—Patients with a diagnosis of gingivitis or periodontitis who required periodontal surgery as part of the therapy were used as subjects. The dressing that contained the hydrogenated fat was the experimental dressing, while the one containing the same powder mixed with eugenol and sweet almond oil in place of the ointment was used as a control. As a general rule, all dressings were removed at the end of one week and were replaced for an additional week. In a few cases, however, it was not necessary to replace the dressing after the first week, and in a few instances it was necessary to replace the dressing again after the second week. No pack was left on for more than a total of three weeks.

The cases were divided into the following two categories:

1. Those in which there was extensive exposure of alveolar bone resulting from mucogingival surgery. The area of exposed alveolar bone involved a minimum of five teeth, but usually six teeth were involved. There were seventy-five cases in this category, fifty of which were controlled as follows: After the periodontal surgery was completed, the control dressing containing eugenol was placed over one-half the exposed alveolar bone, while the experimental dressing was placed over the other half.

2. Cases in which there was a moderate exposure of alveolar bone following osteoplasty. The area of bone exposed was always less than five teeth; usually one or two teeth were involved. In this category there were 360 cases treated with the experimental pack and 300 cases treated with control pack.

In all cases of osseous surgery, an effort was made to leave the periosteum intact.

Findings.—

1. No allergic reactions occurred in either the experimental or the control patients.

2. No candida infection was seen in either group. These above observations are based on a total of 735 cases in which zinc bacitracin was incorporated in the powder.

Fig. 1.—One week postoperatively. In 25 per cent of the cases the granulation tissue under the experimental dressing (arrows) was smoother and not as red in appearance as the control (to left of arrows).

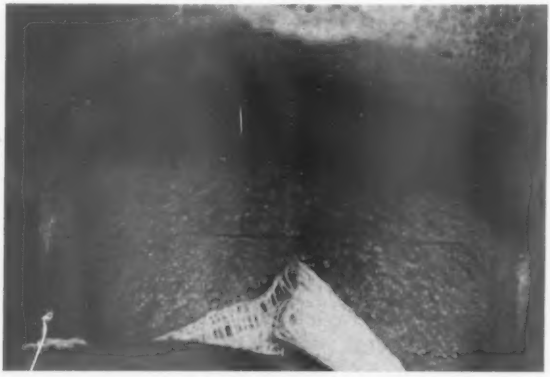

Fig. 2.—A dark, unilateral discoloration of the tongue (arrows) was observed in three cases. This discoloration occurred on the side in which the dressing was placed.

3. The experimental dressing was much easier to handle and adhered to the teeth better than the control.

4. The eugenol dressing caused some immediate discomfort, such as a slight burning sensation and a eugenol taste in all cases, while the experimental dressing caused none of these unpleasant sensations.

5. In one-fourth of the cases involving extensive exposure of alveolar bone, the healing seemed to be at a more advanced stage at the end of the first week,

that is, the granulation tissue was smoother and not as red and granular in appearance as the tissue under the control pack (Fig. 1). At the end of the second week, the healing was about the same under both dressings.

6. When the dressing is applied immediately after mucogingival surgery, it is not necessary to use an extremely stiff mixture in order to obtain good results.

7. The eugenol pack developed sharp, annoying edges in approximately 5 per cent of our controls. The experimental dressing did not develop these hard, sharp edges at any time.

8. In the limited number of cases in which the experimental dressing was placed against an acrylic facing, no pitting of the acrylic occurred.

9. In three cases out of a total of 435 in which the experimental dressing was used a unilateral discoloration occurred on the side of the tongue with the dressing (Fig. 2). Smears from these areas were negative for fungi. The discolorations disappeared promptly upon removal of the pack and were asymptomatic.

DISCUSSION

In view of the frequency with which periodontal dressings are used, the lack of experimental work in this field seems unusual. In a previous publication we reported and commented upon the few available publications that we were able to find.[1]

Lack of allergic manifestations from the topical use of zinc bacitracin during the past two years of clinical trial demonstrates the safety of the antibiotic. During this entire period only one case of candida infection occurred, and this was reported by us previously.[1] The three cases of unilateral tongue discoloration represent a new and unexplainable finding. The patients were unaware of the condition, and since it promptly and completely disappeared upon removal of the pack it is not considered a significant complication.

While either acrylic stents or extremely stiff mixtures of dressing have been recommended following mucogingival surgery,[2] we have not found stiffness a necessary property in our dressing. Actually, our pack does not set very hard at any time, and yet we have been able to obtain good results following all types of periodontal surgery.

The elimination of a foul odor and taste by a dressing which contains zinc bacitracin was further substantiated in this investigation.

The powder portion of our present periodontal pack is a slightly modified version of our previous formula. The powdered Gelfoam, formerly incorporated for its hemostasis, has been omitted. Clinical investigation has shown that in the amounts used it was of doubtful value.

CONCLUSIONS

A dressing consisting of a powder containing zinc oxide, rosin, and 3,000 units of zinc bacitracin per gram, mixed with an ointment of zinc oxide and

hydrogenated fat, is an effective postgingivectomy pack. It is easier to use, is more comfortable to the patient, and appears to be better tolerated in the first postoperative week than one which contains eugenol.

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THE RESPONSE OF SYSTOLIC AND DIASTOLIC BLOOD PRESSURES TO DENTAL STRESS

Irwin I. Ship, D.M.D., Bethesda, Md.

IN 1958 Cheraskin and Prasertsuntarasai[1-4] found significant blood pressure changes prior to and following dental extractions in normotensive and hypertensive adult subjects. A statistically significant mean rise of 29 mm. Hg systolic pressure was noted in nonsedated hypertensive patients, and a 14 mm. Hg systolic pressure rise was seen in sedated normotensive subjects when extractions were begun. Neither group received epinephrine in the local anesthetic solutions. A single blood pressure determination was obtained from each patient before, during, and after the procedure. However, ambient fluctuations of the blood pressure have been found when frequent time-sequence blood pressure determinations were obtained. Landis and Gulette[5] took blood pressure readings at fifteen-second intervals in twenty-five subjects over periods of from fifteen to 120 minutes. Changes of 8 to 10 mm. Hg within one minute were common in unemotional situations, while changes as high as 18 to 20 mm. Hg within one minute occurred in emotional situations. It was felt, therefore, that a minute-to-minute recording system for a study of the blood pressure responses to various types of dental treatment would be more meaningful.

MATERIALS AND METHODS

This study was performed on ten male and seven female patients whose ages varied from 19 to 42 years, with a mean age of 30 years. The patients, who were seen in the Dental Clinic, Clinical Center, National Institutes of Health, were in good health during the study, and in each case a carefully taken history revealed no previous cardiovascular or renal disease.

After arrival at the dental clinic, each patient was taken to an isolated room in which a single dental assistant and I performed all dental and control procedures. All operative procedures were performed with the same dental engine, calibrated at 15,000 r.p.m., and an air-water spray was used. In all oral surgical procedures 2 per cent lidocaine containing no epinephrine was used for anesthesia.

From the Clinical Investigations Branch, National Institute of Dental Research, National Institutes of Health, United States Public Health Service, Department of Health, Education, and Welfare.

Minute-to-minute blood pressure determinations were automatically recorded with the Colson Model 110 blood pressure recorder (Fig. 1). This unit was modified by the addition of an impulse recorder for marking the initiation and completion of a procedure on the chart. The recorder was manually calibrated before and after each appointment. Correction factors from 0 to 4 mm. Hg were found in individual studies. The factor remained constant throughout each appointment, however, and no adjustments were necessary. The unit functioned quietly out of sight of both the investigator and the patient. The angulation of the patient in the dental chair was maintained at 55 degrees from the horizontal in order to minimize postural blood pressure variations.

Fig. 1.—The Colson Model 110 blood pressure recorder prior to calibration in the dental clinic.

The dental appointments were divided into four categories:

1. Control appointments. The patient remained at rest in the chair throughout the appointment but was unaware that no procedure was to be performed. The dentist was not present, and there was no therapy.
2. Operative dentistry I (excavation and restoration of carious teeth).
3. Operative dentistry II (polishing of restorations).
4. Oral surgery (extraction of teeth).

All appointments were for a period of forty minutes. Several minutes were required for positioning the patient and adjusting the blood pressure recording unit. No less than thirty consecutive systolic and diastolic blood pressure determinations were obtained during a single visit. The appointment categories

were selected at random, and the appointments were varied as to time of day and day of the week by means of random number sequence tables.*

For the purpose of analysis, each appointment was divided into three periods. During the first period the subject sat at rest in the chair for five to ten minutes, unaware of whether or not a procedure was to be performed. During the second period the various procedures were performed, and during the third period the subject sat at rest in the chair for five to twenty minutes. For control visits, of which the patient had no prior knowledge, these subdivisions were not necessary.

RESULTS

A total of sixty appointments were recorded during the course of this study. Due to the random selection of the treatment categories, the sequence varied for each patient. Not all patients were studied in each category. There were sixteen control appointments, twenty-four appointments for operative dentistry I, ten for operative dentistry II, and ten for oral surgery.

Surprisingly, when the mean systolic or diastolic pressures of each group were examined, no significant variations were found (Table I). Age, sex, and initial systolic or diastolic pressures had no effect on the magnitude of mean fluctuation experienced. Time of day, day of the week, and the specific nature of the procedure also had no effect. When control periods were compared with the most traumatic procedures (oral surgery), a mean difference of only 8 mm. systolic and 1 mm. diastolic pressure was noted. Operative dentistry revealed a change of 4 mm. systolic and 3 mm. diastolic, while polishing of restorations was associated with a pressure change of 1 mm. systolic and 2 mm. diastolic. The mean blood pressures, however, when examined individually, showed significant differences in range and occasionally a more marked variation in the mean (Tables II and III). Furthermore, when individual blood pressure charts were examined, the patients appeared to fall into three response patterns:

1. *Nonreactor.* Six patients demonstrated minimal changes in systolic and diastolic blood pressures not related to the procedure or appointment sequence. Essentially, the pattern for control appointments was duplicated in all other procedures (Fig. 2).

2. *Reactor.* Eight subjects responded to various procedures with moderate fluctuations of blood pressure. Both systolic and diastolic blood pressures rose and fell during the procedure, but they usually stabilized when the operative procedure was terminated. Whereas a level line was seen during control appointments, moderate changes were noted when any procedure was attempted (Fig. 3). A moderation of the level of initial response was frequently seen in later appointments.

*The random number sequence guide was prepared by Mr. Carl L. White, Statistician, National Institute of Dental Research, National Institutes of Health, Department of Health, Education, and Welfare, Bethesda 14, Maryland.

TABLE I. MEAN BLOOD PRESSURES BY INDIVIDUAL PROCEDURES

PROCEDURE	NO.	ARRIVAL CONTROL (1)		DENTAL PROCEDURE (2)		CHANGE (1-2)		FINAL CONTROL (3)		CHANGE (2-3)		CHANGE (1-3)	
		SYS-TOLIC	DIAS-TOLIC	SYS-TOLIC	DIAS-TOLIC	SYS-TOLIC	DIAS-TOLIC	SYS-TOLIC	DIAS-TOLIC	SYS-TOLIC	DIAS-TOLIC	SYS-TOLIC	DIAS-TOLIC
Operative dentistry	224	109	72	114	79	+5	+7	111	78	−3	−1	+2	+6
Polish	10	112	76	114	82	+2	+6	112	80	−2	−2	0	+4
Oral surgery	10	105	72	114	77	+9	+5	109	76	−5	−1	+4	+4
Control	52	110	74	111	78	+1	+4	111	78	0	0	+1	+4

TABLE II. MEAN AND RANGE OF SYSTOLIC BLOOD PRESSURES OF INDIVIDUAL PATIENTS BY PROCEDURE

PATIENT	AGE	SEX	ARRIVAL CONTROL		OPERATIVE DENTISTRY		POLISHING		ORAL SURGERY		FINAL CONTROL	
			M	RANGE	M	RANGE	M	RANGE	M	RANGE	M	RANGE
1	19	F	93	80-107	93	90- 99					92	80-111
2	28	F	89	86-100	96	85-110					90	85- 97
3	28	F	106	90-125					108	62-135	114	90-140
4	30	F	94	80-103	92	79-105			94	88-100	94	89-101
5	31	F	98	80-111			103	95-113	126	87-127	97	94-115
6	34	F	101	92-117	104	93-118	106	92-128			93	88- 99
7	36	F	109	102-126							113	99-127
8	23	M	116	105-130	122	117-127			127	110-129	115	110-124
9	26	M	120	114-125			121	105-127			119	108-130
10	28	M	109	92-120	116	100-131	113	107-118			106	90-127
11	28	M	119	113-126	130	125-140			133	115-167	130	124-142
12	29	M	105	92-120	116	106-127			120	108-125	109	90-124
13	32	M	113	97-128	114	103-120					113	99-127
14	32	M	109	103-120							112	102-127
15	34	M	117	115-124			117	99-125			117	108-125
16	34	M	118	108-128	123	113-130	126	120-132			118	108-137
17	42	M	128	117-148	131	120-144					132	117-150

O.S., O.M. & O.P. April, 1960

TABLE III. MEAN AND RANGE OF DIASTOLIC BLOOD PRESSURES OF INDIVIDUAL PATIENTS BY PROCEDURE

PATIENT	AGE	SEX	ARRIVAL CONTROL		OPERATIVE DENTISTRY		POLISHING		ORAL SURGERY		FINAL CONTROL	
			M	RANGE	M	RANGE	M	RANGE	M	RANGE	M	RANGE
1	19	F	66	64- 82	74	67- 76					77	70- 86
2	28	F	72	52- 75	73	62- 81					76	70- 85
3	28	F	71	60- 83					59	35- 85	73	38- 91
4	30	F	61	48- 67	64	55- 81			66	64- 70	65	45- 75
5	31	F	78	65- 85			79	62- 87	92	85- 99	75	70- 85
6	34	F	63	56- 67	67	57- 75	72	48- 80			66	58- 73
7	36	F	83	47-100					86	45- 90	85	50- 95
8	23	M	68	47- 72	76	70- 86			75	70- 77	74	62- 87
9	26	M	76	74- 84			85	75-105			85	47- 90
10	28	M	66	50- 77	67	52- 85	64	50- 75			70	60- 80
11	28	M	81	68- 95	92	70-105			94	68-110	93	68-105
12	29	M	72	60- 94	83	67- 97			96	94-102	79	68-100
13	32	M	72	52- 76	78	72- 85					75	62- 84
14	32	M	63	42- 74							68	60- 78
15	34	M	77	68- 91			82	67- 90			78	60- 91
16	34	M	68	60- 89	86	60-103	93	85- 97			81	63- 92
17	42	M	101	90-125	106	90-125					105	87-124

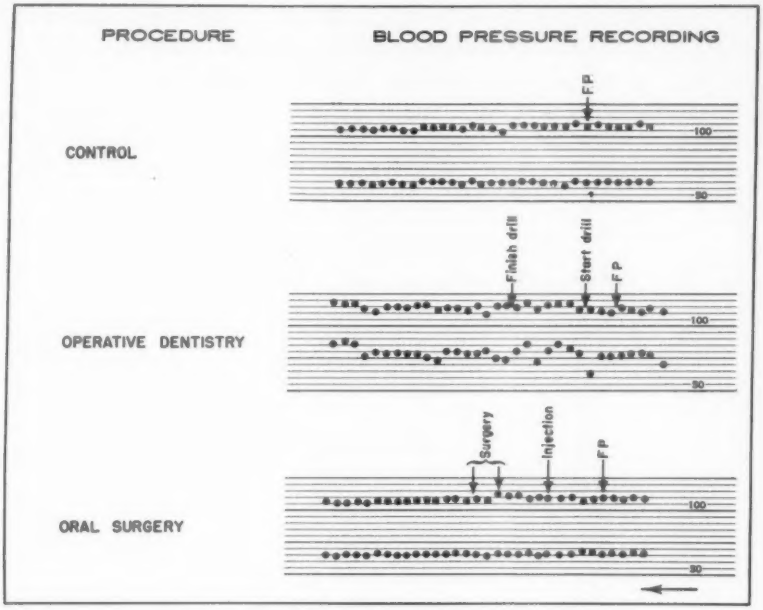

Fig. 2.—Nonreactor.

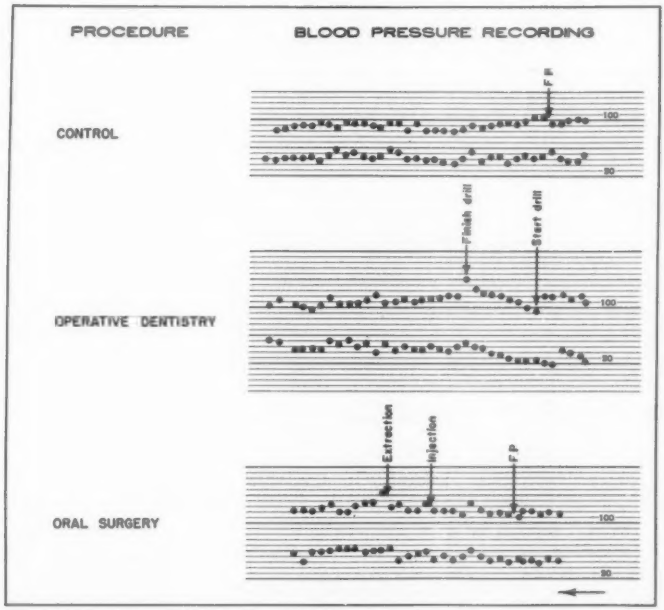

Fig. 3.—Reactor.

3. *Hyperreactor.* Regardless of the procedure, three patients responded with large fluctuations in both systolic and diastolic blood pressures. Pressures increased, in two patients, while a decrease in blood pressure was seen in the third. Often the blood pressure recording unit was unable to secure consecutive recordings due to the rapid fluctuations* (Fig. 4).

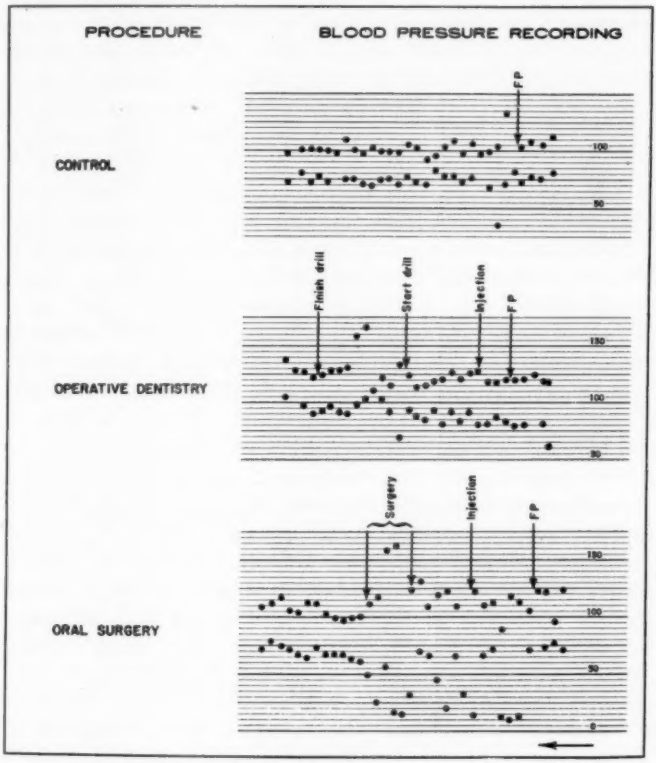

Fig. 4.—Hyperreactor.

A finger prick was usually done by the technician during the first phase of each appointment. The blood pressure response to this slightly painful standardized procedure gave a fairly accurate indication of the nature of the patient's response to the dental procedure.

DISCUSSION OF RESULTS

In recent years increased attention has been given to the physiologic effects that various stress situations have on the human organism. During this period

*Since the unit was adjusted to record at one-minute intervals, if an accurate pressure could not be determined in forty-five seconds, the unit failed to record.

the physiologic responses to various phases of dental therapy have been documented. In 1957 A. G. Ship[6] demonstrated significant eosinopenia, suggesting an increased adrenal steroid secretion, in four adult subjects following oral surgery. In a broader series, significant eosinopenia was noted after a variety of dental procedures.[7]

The purpose of this study was to investigate the effects of dental procedures on the blood pressure of healthy adult patients. That the maintenance of systolic and diastolic blood pressures is an extremely labile phenomenon is substantiated by the data presented, and minute-to-minute changes do occur in the healthy human adult. Fluctuations in both systolic and diastolic blood pressures during control and dental situations were great, so that mean elevations or depressions tended to lose significance when procedures or time periods were compared. Six patients were classified as "nonreactors" who, for the most part, were calm and indifferent to the dental environment. Their interest in the dental appointment appeared to be minimal, and their patterns did not differ from one appointment to the next. Eight patients were classified as "reactors." Patients in this group were concerned with their teeth and with dentistry. Their patterns differed from procedure to procedure, and clear contrasts were observed when control records were compared with other categories. Three patients were classified as "hyperreactors" on the basis of their blood pressure responses to dentistry. Fluctuations in both systolic and diastolic pressures often were rapid and severe. These subjects appeared to be tense and apprehensive prior to and during every dental appointment.

It is interesting to note that analyses of group changes tend to obscure individual response patterns. Averages did not recognize individual responsiveness to dentistry and the dental environment generally. The finger prick, a common procedure that is familiar to the medical patient, provoked blood pressure responses comparable to the responses elicited by more extensive dental procedures, indicating that blood pressure reactions to many stresses are similar and are not necessarily related to the magnitude of the stress as interpreted by the dentist. The patient's response to such procedures as venipuncture and finger prick may therefore provide an indicator of the blood pressure responsiveness to be expected during dental procedures. It appears that the dentist subjects his patients to procedures affecting the blood pressure which are comparable with other life situations and that he need be concerned only with certain hyperresponsive patients.

SUMMARY AND CONCLUSIONS

The effects of dental procedures and control appointments on the blood pressure were investigated in seventeen healthy adult subjects. Analysis of results revealed the following:

1. The net differences in either mean systolic or mean diastolic blood pressures between procedures were insignificant.

2. Patients appeared to fall into three categories of response to dental procedures and were classified as "nonreactors," "reactors," and "hyperreactors."

3. The relative importance of expected blood pressure changes in the patient undergoing dental treatment has been discussed.

The author wishes to acknowledge the valuable assistance of Miss Virginia Moore, Miss Mary Alice Case, and Dr. A. Donald Merritt in this project.

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ANNOUNCEMENTS

University of Minnesota

The School of Dentistry announces the availability of a limited number of United States Public Health Service Fellowships leading to a Ph.D. degree in a basic science, such as pathology, bacteriology, physiology, physiological chemistry, and anatomy. The minor will be taken in a dental clinical field. The programs are designed for those planning to pursue a teaching and/or research career. The stipend is $5,000.00, tax free. Tuition is included.

For further particulars, contact The Office of the Dean, School of Dentistry, University of Minnesota, Minneapolis, Minnesota.

University of Washington
Graduate Program in Dentistry

The degree of Master of Science (M.S.D.) is awarded for satisfactory performance in the academic and clinical aspects of the discipline and the submission of a satisfactory thesis reporting on research in basic science. The following courses are available to properly qualified applicants:

Fixed Partial Dentures. Executive officer: K. N. Morrison, D.D.S., M.S.
A minimum of three consecutive quarters (nine months) of residence is required for a major. Class limited to three.

Operative Dentistry. Executive officer: Gerald D. Stibbs, B.S., D.M.D.
Three quarters (nine months). Class limited to three.

Oral Pathology. Executive officer: Leo M. Sreebny, A.B., D.D.S., M.S., Ph.D.
Eight quarters (twenty-four months). Class limited to two.

Oral Surgery. Executive officer: John D. Gehrig, D.D.S., M.S.D.
Three quarters (nine months) plus two-year hospital residency, combined academic and hospital work. Limited to one.

Orthodontics. Executive officer: Alton W. Moore, D.D.S., M.S.
Six quarters (eighteen months). Class limited to ten.

Pedodontics. Executive officer: David B. Law, B.S.D., D.D.S.
Five quarters (fifteen months). Class limited to two.

Periodontics—Endodontics. Executive officer: John I. Ingle, D.D.S., M.S.D.
Eight quarters (twenty-four months). Limited to five students in periodontics and three in endodontics. The course in periodontics is given by Dr. Saul Schluger.

Prosthodontics. Executive officer: Harry A. Young, D.D.S.
Three quarters (nine months). Class limited to two.

Tufts University

Dr. Charles E. Stuart of California, noted authority in the field of occlusion, will be principal lecturer at the Tufts eleventh annual Berkshire Conference in Oral Pathology and

Periodontology to be held June 19 to 23, 1960, in Lenox, Massachusetts. Dr. Stuart will devote two days to the following subjects:

Principles and Use of Diagnostic Instruments for Instituting a Therapeutic Occlusion.

Principles and Objectives of Therapeutic Occlusion.

The Technique for Restoring Occlusion for Patients With Periodontal Problems.

Albert Einstein Medical Center

The Department of Postgraduate Medical Education announces that a course entitled "Dentistry for the Handicapped Child" will be given by Morris Kelner, D.D.S., and associates on April 25 to 27, 1960. The course is designed to enable the dentist to render adequate and proper treatment for the handicapped child. Instruction will provide a fuller understanding of the pedodontic, pediatric, and psychologic factors as they affect the dental problem of the handicapped child.

The lectures will include a background of physical, mental, and emotional handicapping conditions; their oral manifestations; modifications in approach to chair treatment; modifications of techniques and methods of rendering adequate treatment; preparation of the patient and operator for total treatment under a general anesthetic in the operating room; periodontal, prosthetic, orthodontic, and speech problems; and psychosomatic and medicodental pediatric problems.

The course will be given at the Northern Division of the Albert Einstein Medical Center in Philadelphia, Pennsylvania. Registration, which is limited to ten students, will close April 10, 1960. The fee for the course will be $100.00.

Boston University School of Medicine

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University and the Massachusetts Memorial Hospitals, announces the following postgraduate courses:

Periodontal Therapy. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Jack Bloom, and Associates. April 4 to 9, 1960. $175.00.

Apicoectomy—Surgical Endodontic Procedures. Herbert Schilder and Leonard Parris. April 18 to 22, 1960. $175.00.

Periodontal Pathology. Henry M. Goldman and Jack Bloom. April 25 to 30, 1960. $250.00.

Pulp Protection in Operative Dentistry. Harold Berk. May 12 to 14, 1960. $100.00.

Periodontal Prosthesis. Leo Talkov, David J. Baraban, Henry M. Goldman, Bernard S. Chaikin, and Gerald M. Kramer. May 16 to 20, 1960. $200.00.

Surgical Procedures in Periodontal Therapy. Lewis Fox and Associates. May 23 to 27, 1960. $200.00.

For further information, write to Director of Post-Graduate Studies, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Massachusetts.

Georgetown University

A postgraduate course in intravenous anesthesia for the dental office will be given by Drs. Harold Krogh, Washington, D. C., and Edward Driscoll, Long Beach, California, Monday through Friday, May 22 to 28, 1960, from 9 A.M. to 5 P.M. Participation is limited

to dentists in the exclusive practice of oral surgery. Enrollment is limited to fifteen, and the deadline for the filing of applications is May 1, 1960.

Application blanks may be obtained by writing to the Registrar, Georgetown University School of Dentistry, 3900 Reservoir Rd., N.W., Washington 7, D. C.

University of Washington

The School of Dentistry, Department of Postgraduate Dental Education, will present a refresher course in exodontia and minor oral surgery May 23 to 27, 1960.

This five-day course is specifically designed to augment the knowledge and techniques of the general practitioner of dentistry. It will include the basic fundamentals of good surgery and the present concepts of minor oral surgical techniques. The daily program will consist of lectures, practical demonstrations, and clinical practice by the participants. Several lectures will be devoted to the medical aspects of dental surgery, including internal medicine, antibiotic therapy, analgesics, sedatives and tranquilizers, blood dyscrasias, and malignant tumors.

Registration for those selected will begin at 8:30 A.M., Monday, May 23, 1960, in Room B-323 of the Health Sciences Building. Tuition for the course will be $165.00. A deposit of $85.00 should accompany each application. All supplies, with the exception of gowns, will be furnished by the department.

Michael Reese Hospital

Dr. A. Martin Schwarz of Vienna will give two courses, one for the orthodontist and one for the general practitioner, on May 2 to 6 and May 9 to 13, respectively.

The course for orthodontists will offer the American orthodontist a rare opportunity to add to his knowledge of cephalometric analysis as well as demonstrate the extensive therapeutic possibilities offered by the removable orthodontic appliance.

In the course for the general practitioner, Dr. Schwarz will demonstrate methods of intercepting and preventing incipient malocclusions with removable appliances.

For complete information, write to Dental Department, Michael Reese Hospital and Medical Center, 29th Street and Ellis Ave., Chicago 16, Illinois.

REVIEWS OF THE LITERATURE

REVIEWS OF NEW BOOKS

Surgery of the Oral Cavity and the Technique of Controlled Tooth Division. W. W. Cogswell, D.D.S., Visiting Professor at Kansas City School of Dentistry, Consultant to the U. S. Air Force Academy, and Oral Surgery Consultant to Fitzsimons Army Hospital. Baltimore, Williams & Wilkins Company; Palo Alto, J. W. Stacey, Inc. Price, $166.00.

Surgery of the Oral Cavity is offered only as a complete unit. It includes a two-volume text, 200 View-Master seven scene reels, an illuminated focusing viewer with transformer and cord set, a film brush, and indexed file for reels, and a fitted carrying case.

Complete *Surgery of the Oral Cavity* kit.

This stereographic presentation, which correlates 1,400 three-dimensional color pictures with a two-volume text, covers the fifty-seven surgical problems most likely to be encountered in practice. A special 3-D projector is available for group showings. The text constitutes a commentary which is keyed by paragraph number to each of the 1,400 pictures. Superbly executed case models of each surgical step are re-created in three-dimensional color pictures for study. The use of realistic case models assures unimpaired viewing of each condition

and procedure. The combination of the three-dimensional pictures and the author's commentary, when examined together, closely duplicates the atmosphere of a clinical seminar.

The stereoscopic pictures are mounted in reels of seven pairs each and are studied through the electrically illuminated View-Master focusing stereoscope, which is held in the hand. The operations covered vary from extractions of teeth by the controlled tooth-division method to removal of foreign bodies from the sinuses and removal of small tumors and cysts. This type of presentation offers classroom usefulness as well as an opportunity for the private practitioner to study the technique with a view to improving his services to his patients.

K. H. T.